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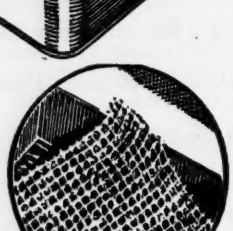

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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—36TH YEAR. SYDNEY, SATURDAY, FEBRUARY 26, 1949. No. 9.

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An Address.[1]

By B. L. W. CLARKE,
President, Queensland Branch of the British Medical Association.

IT is about thirty years since I joined the British Medical Association and at that time I did not think that one day you would elect me Branch President. I am deeply conscious of the honour and thank you most sincerely. When I review the long line of my predecessors it is with a certain amount of trepidation that I occupy this chair, because amongst them are many illustrious men, who have not only been distinguished in their own particular branch of medicine, but have done a great deal for their fellow members, and much to enhance the prestige of our profession. It is my hope that when my term expires I can pass on to my successor that same unsullied record of service that has been given in the past.

During the last quarter of a century a great many changes have taken place in both the scientific and political aspects of medicine, and these changes, particularly those in the latter sphere, have considerably altered and increased the activities of the British Medical Association. Until recently the greater portion of our activity has been of a scientific nature, but there was to a certain extent some semi-political activity in the nature of collective bargaining in order to make arrangements satisfactory to the Branch and the State Department of Health and Home Affairs, and other organizations such as the friendly societies *et cetera*. In all of these negotiations there has rarely arisen any serious obstacle to prevent a solution satisfactory to both parties. In fact our negotiations with the State Government have usually been most harmonious. During the last year the British Medical Association has become more prominent in medico-political activities and it looks as though there will be further increase in the impending future.

On the scientific side many great advances have been made which have revolutionized diagnosis and therapy. Some of these measures have resulted from the careful observations of an individual doctor, who when he was satisfied that his "discovery" was beneficial, has passed on his information in order that others may put it into practice and confirm its usefulness. It has always been a code of honour amongst doctors that no beneficial remedy should remain a secret or be commercialized for personal profit.

Perhaps there may be a lighter side to this, and possibly for our own protection, because doctors as a whole are regarded as notoriously bad business men, and prior to the recent world war seemed to have a number one priority to invest moneys in any "wild cat" scheme.

The scientific advancement has altered the course of many types of disease; for instance, the "sulpha" drugs and penicillin, used in their proper sphere, cut short many illnesses. Today we do not see so much of the ravages of osteomyelitis as in the pre-"sulpha" days; and this has had its effect on the radiologist, who before the use of these drugs was constantly encountering bone infections in their various stages, but today they are far less common and one has to fall back on the old films to demonstrate the many changes. The control and treatment of venereal disease is another example, aneurysms and Charcot's joints are comparative rarities in our hospitals.

An example of the application of physics to medicine is illustrated by the progress of X-ray and radium therapy. There is certainly a great deal yet to be learnt, but at least one gratifying result is the response of skin cancer to this method of treatment.

Many of these advancements cost considerable sums of money and treatment may be beyond the financial ability of a large section of the public, but in order to overcome this difficulty and not deprive the sick of necessary treatment, there are the large public hospitals which are either

[1] Read at the annual meeting of the Queensland Branch of the British Medical Association on December 10, 1948.

heavily subsidized or fully financed by the Government. So that there is no reason whatever, at the present time, why any individual, no matter what his status, should be deprived of treatment. The various governments have not been idle in matters affecting public health. In preventive medicine we are all familiar with the results obtained by anti-typhoid inoculation and diphtheria immunization; while other institutes which have given invaluable service to the public are the Commonwealth Serum Laboratories, the Commonwealth X-Ray and Radium Laboratory *et cetera*. In the past, with the exceptions of certain necessary restrictions, the individual doctor has been free to treat his patient as he has thought fit.

Our increasing knowledge has necessitated specialization. It is not possible for the individual to keep abreast of all advances, so some members of our profession have concentrated their studies along certain avenues, and eventually have become specialists in a particular branch of medicine. The law of this State requires the registration of specialists and this can come about only by the fulfilment of certain standards and qualifications.

The specialist occupies an important place; in fact, although I say it myself, the profession in general would be placed at a considerable disadvantage if deprived of the assistance of the radiologist. I do not intend to suggest that he is always correct in his interpretation.

Specialist training is time consuming and costs money, yet curiously enough our taxation laws regard the cost of this extra study as expenditure of capital and allow no deduction for income tax purposes. If one obtains degrees or diplomas in order to comply with the requirements of the law, one must therefore do it at one's own expense.

It is hoped that in these days when there is this tendency to specialization, the position of the general practitioner will not be overlooked. In my humble opinion the general practitioner is the backbone of the profession; he is not only the family medical attendant, but he is in so many cases the family adviser, and his opinion is sought on matters not altogether medical, and the patient knows quite well that this confidence will be respected. The general practitioner service in this country has reached a very high standard of efficiency, and it would be tantamount to a national disaster, I consider, if this service and the doctor-patient relationship should be disturbed. Probably no other branch of the community works harder. The general practitioner does not know a 40-hour week—nor does his wife.

We are now confronted with what may be a revolutionary change for the whole medical profession by the introduction into Parliament recently of the *National Health Service Bill*. We all realize that, with the present trend of world affairs, medicine will not be excluded, and it is our hope that the impending changes will be evolutionary rather than revolutionary and that the doctor-patient relationship will never be disturbed.

At the present time we are not conversant with full details, nor how the Act will eventually affect us and our patients.

On the surface there are some advantages to both parties. A schedule of fees is to be drawn up and the Government promises to pay 50% of them. This may be a good thing for doctors and patients, and it has been suggested that it will reduce our "bad debts". Many of the "bad debts" are not always the fault of the patient.

In a scheme which is partly or wholly free, provision should be made to guard against the cluttering up of doctors' consulting rooms by patients requiring unnecessary treatment, as this leads to overwork for the doctor, and acts to the detriment of those who are really in need of medical assistance. In all negotiations between the British Medical Association and the Federal Government, we should strongly advocate that the patient should have an absolute right to select his own doctor, that the doctor should be free to practise where and when he likes, that the doctor-patient relationship should not be undermined, and that all agreements should be between the Government and the British Medical Association and not with individual doctors.

If the new legislation should be of such a nature that it can be merely altered by regulations at will, I can foresee that the time will soon come when we will be regimented, and as Lord Horder recently said: "The profession will become a branch of the civil service."

A recent American review stated that Mr. Bevan, Britain's Health Minister, was somewhat embarrassed by the public's enthusiasm for government-financed doctoring. He said: "The health moves will fail unless the people use it intelligently, sparingly, and prudently."

As the provisions of the new bill are now the subject of negotiation between the British Medical Association and the Government it would be out of place for me to discuss this further.

The general body of practitioners has been wise in the selection of its councillors and I feel sure that you can confidently rely on them to watch over the interests of the profession and of our patients.

PRIMARY ATYPICAL PNEUMONIA: A REPORT ON FOURTEEN CASES OBSERVED AT THE ROYAL ADELAIDE HOSPITAL.

By P. W. VERCO,[1]

From the Institute of Medical and Veterinary Science, Adelaide, and the Department of Experimental Medicine, University of Adelaide.

"PRIMARY atypical pneumonia, ætiology unknown" is one of many terms used to designate an acute respiratory infection prevalent in America and apparently of widespread distribution. It has been much more widely recognized since the advent of the sulphonamide drugs and their success in the chemotherapy of the bacterial pneumonias. It is characterized by an insidious onset with constitutional symptoms of malaise (including general aching and pain in muscles or joints), chilliness, fever, anorexia and headache, commonly frontal. Local symptoms referable to the nose or throat are frequent at the onset. The constitutional symptoms become more severe, to be followed shortly by an unproductive, irritating cough.

The illness varies greatly in its course, severity and duration. In its lesser forms pulmonary changes often are not detected by clinical means, but are revealed by X-ray examination of the chest, and recovery may follow at this stage. Symptoms may increase in intensity, and the cough, now productive of a viscid, mucoid sputum, becomes frequent and distressing; at times it occurs in paroxysms and causes exacerbation of the headache. Fever continues, of variable type, and profuse sweating may occur; the pulse is rapid and full, and the respiration rate is increased in the more severe forms. On the other hand, in many cases in which radiological evidence of consolidation is obtained, the respiration and pulse rates are not altered. The pyrexia and the pulse and respiration rates, in the more severe forms of the illness, are an unexpected contrast to the apparent well-being and mental clarity of the patient. Chest pain, when present, is usually a sense of discomfort behind the sternum or along the costal margin, and severe pleural pain is rare. The sputum is occasionally blood-stained; cyanosis may accompany the more severe type of illness.

Signs of pulmonary involvement are most commonly râles, with some dulness to percussion, and these vary considerably in extent, but often are less evident than would be supposed from radiographic examination. Polymorphonuclear leucocytosis, absent in the initial stages, may appear as the disease progresses. In the serum of a proportion of patients cold agglutinins can be detected during the latter part of the illness and in convalescence.

This condition runs a self-limiting course, apparently not influenced by therapy with sulphonamides or penicillin, the pyrexia subsiding by lysis with gradual improvement in the patient's condition. Convalescence is frequently

[1] This work was done with the aid of a grant from the National Health and Medical Research Council.

prolonged with asthenia, and is often accompanied by persistence of a mild cough and delay in resolution of the pulmonary lesion evident by serial radiographic examination.

Complications are uncommon and the mortality rate is ordinarily stated to be low.[1][2]

Historical Review.

During the last century the conception of acute inflammatory diseases of the lung has altered materially. Although clinical and morbid anatomical observations have contributed greatly to knowledge of this subject, they have been largely overshadowed by discoveries of many of the causal agents of this group of diseases. Differentiation between lobar pneumonia, interstitial pneumonia and bronchopneumonia, of the era of the morbid anatomists, was little affected by the association of the pneumococcus with pneumonia. Later refinements in bacteriology and immunology disclosed that hæmolytic streptococci and staphylococci, Friedländer's bacillus and *Hæmophilus influenzæ* were also sometimes responsible for acute inflammation of the lungs. The typing of pneumococci led to the important generalization that the course and prognosis of pneumococcal pneumonia depended largely on the type of pneumococcus.

The great variation in the bacteria associated with pneumonia complicating many cases of measles in the epidemics of 1914-1918 recurred in the influenza pandemic of 1918, in which the bacteria isolated were often heterogenerous and differed greatly from time to time and from place to place. There was also, in the influenza pandemic, great variation in the course of the illness and of the anatomical change found *post mortem*. Among the latter, in some cases the pulmonary lesions found were strikingly similar to those found after death from inhalation of the irritant gases during the first World War.

The confusion created by the absence of a constantly responsible bacterium stressed the need for the isolation of the common ætiological agent of this obvious pandemic. Increasing interest in viruses and improvements in technique were not fruitful until 1933, when the ætiology of epidemic influenza was definitely established by transmission to ferrets of influenza A virus. This finding led to analysis of the considerable experimental work already carried out on the production of pneumonia in animals by bacteria, by viruses, or by both. Comparison of these findings, particularly with material obtained from human subjects who died in the pandemic, and of those in other known or suspected virus disease in which the lung had been found bacteriologically sterile *post mortem*, yielded information not only as to which lesions were primarily due to virus and which lesions were due to superimposed infection with bacteria, but also as to the characteristic clinical behaviour of "pure" infection with specific bacteria. Subsequent study of minor epidemics of influenza led to the discovery of influenza B virus. The introduction of a hæmagglutination test by Hirst has provided a useful serological method of detecting infection among the population by a strain of either virus and facilitated the detection of epidemics of influenza.

It is generally agreed that the character of pneumonia has changed perceptibly over the last decade; there has been a considerable and real decrease in the incidence of lobar pneumonia. Whether this entirely is due to the advent of the sulphonamides and penicillin, which have proved to be specific therapeutic agents for pneumonia due to the pyogenic cocci in almost all cases is not known. The general success of these drugs has thrown into prominence a group of cases of pneumonia in which the illness is not affected by chemotherapy. These include a minority in which the bacterium, ordinarily susceptible, may be shown *in vitro* to have lost this susceptibility to a varying degree. From the remainder of this heterogenous group has emerged the more severe type of illness of primary atypical pneumonia.

Improved X-ray equipment has facilitated examination of the thorax, and the more general use of this method of examination, particularly in mass surveys, has frequently revealed transient pulmonary opacities, often not detected by clinical means, causing but little systemic discomfort, and commonly associated with acute disease of the upper part of the respiratory tract.

With the outbreak of the recent war occurred the anticipated epidemics of acute respiratory disease among recruits, which were expected to follow the congregation of young adults in military camps. Routine X-ray examinations of the chest revealed unexpectedly frequent pulmonary involvement, and most investigators failed to incriminate either influenza virus. American observers seized the opportunity to study an unexpectedly high incidence of such an unusual type of pneumonia, occurring among troops at Camp Clairborne, Louisiana, in 1941, and the Commission on Acute Respiratory Diseases was set up. This commission has placed the clinical and laboratory characteristics of primary atypical pneumonia on a sound basis.[3]

The commission was unable to identify or isolate the causal agent; but subsequent experimental transmission to human volunteers by filtered throat washings and sputum has indicated that it is a virus or viruses.[4] In particular the commission's studies established that this entity was not due to infection with the influenza virus, with the psittacosis group of viruses, or with *Rickettsia burneti* of Q fever, although these diseases may occur in epidemic form and closely resemble primary atypical pneumonia in their manifestations. Coccidioidomycosis, in the early phases of which primary atypical pneumonia may be simulated, was also excluded.

Eaton, Meiklejohn and Van Herick were able to transmit to chick embryos and adapt by passage a filterable agent from the sputum or lung tissue of certain subjects of primary atypical pneumonia.[5] Suspensions of infected chick embryo tissues, after intranasal inoculation into cotton rats and hamsters, produced pulmonary lesions which were similar to those seen after instillation of infective human material. The agent was propagated in chick embryos and it was found that it could be specifically neutralized by the serum of patients recovered from primary atypical pneumonia, but not by acute phase serum. These workers stressed that in an epidemic of primary atypical pneumonia, in itself suggesting a single ætiology, a substantial proportion of cases was apparently due to this new virus; but it is unlikely that it is the causative agent of all cases of primary atypical pneumonia.

Although there is a general impression in the large teaching hospitals in Australia that primary atypical pneumonia does occur here, no detailed report of pneumonia in this country has been published since that of Holmes à Court in 1940.[6] This referred to the bacteriology of 1000 consecutive cases of pneumonia and to the response to treatment with sulphapyridine in a smaller series of 60 cases of pneumococcal pneumonia. In the latter series 15 patients failed to respond to therapy within forty-eight hours, in six cases the fever was unaffected by sulphapyridine and five patients died. It is possible that some of these patients were suffering from pneumococcal pneumonia which failed to respond to sulphapyridine and that a few were suffering from primary atypical pneumonia.

In this paper 14 cases, in which the clinical and pathological picture conformed to that of primary atypical pneumonia, are described in detail. These cases, together with 20 other cases which closely resembled them, but which were excluded from the series because of incomplete investigation, represent one-seventh of the cases of pneumonia selected for study from a total of some 1000 cases of acute pulmonary disease treated at the hospital during a period of fourteen months.

Method.

The Royal Adelaide Hospital.

The Royal Adelaide Hospital, in which the patients presented were treated, is a general public hospital of 750 beds, comprising surgical, medical and ancillary services. This hospital provides for patients over the age of twelve years not suffering from acute infectious diseases.

The medical service contains 250 beds and is usually filled to capacity. Patients are drawn from Adelaide, its

suburbs and the neighbouring districts, as the Royal Adelaide Hospital is the sole public hospital available for this area.

Material.

Patients suffering from pneumonia who were admitted to the Royal Adelaide Hospital after June 1, 1946, and up to October 1, 1947, with the omission of December, 1946, and January, 1947, were investigated as set out below.

Certain patients were excluded, as they exhibited factors predisposing them to pulmonary infection or infarction, which would confuse the clinical picture. In particular those subjects who were over the age of sixty years or had a history of recent trauma, surgery or anæsthesia were excluded. Also those subjects who had gross deformity of the thoracic cage, poor physique or general debility, or who were suffering from cardio-vascular or renal disease, neoplasm, hydatid disease, chronic lung disease or tuberculosis, were excluded from this study.

Leucocyte and Differential Leucocyte Counts.

Leucocyte and differential leucocyte counts were performed soon after the patient's admission to hospital, if possible before the administration of sulphonamides or penicillin. Those patients to whom these drugs were administered prior to their admission to hospital are indicated in Table I, as also is the day on which the leucocyte count was performed. Venous blood was usually used.

X-Ray Examination of the Chest.

An X-ray examination of the chest was made as soon as possible after the patient's admission to hospital. It had been hoped that postero-anterior and lateral films would be prepared in every case, with weekly follow-up films; but owing to shortage of films and to intermittent pressure of work in the radiological department, this ideal could not be realized and lateral films were often omitted. The severe illness of the patient and the rapid respirations often resulted in a poor skiagram taken on a portable machine.

Sputum.

Specimens of sputum, when obtainable, were examined in direct smears for pathogenic organisms (Ziehl-Neelsen staining was included) and cultures were attempted, in some cases both aerobically and anaerobically. Penicillinase and para-amino benzoic acid were not incorporated in the media. No effort was made to identify further any Gram-negative bacilli.

Serum.

Specimens of serum were obtained during the acute phase of the illness and in early and in late convalescence. Serum was not inactivated and was stored in sealed glass ampoules at −20° C. without preservatives.

Cold Agglutinins.

A number of cold agglutinin titrations were performed by Mr. R. W. Sheppard on specimens of serum shortly after they had been obtained. The method followed was that of Turner *et alii*,[7] five-day-old cells being used.

Miss P. E. Lind, of the Walter and Eliza Hall Institute, Melbourne, kindly performed cold agglutinin titrations and "T" agglutinin titrations on certain specimens of serum.[8]

Psittacosis Complement Fixation.

In both Melbourne and Adelaide the same strain of psittacosis virus was used as an antigen. The antigen consisted of pooled allantoic fluid, bacteria-free. Such complement fixation tests were carried out by Miss P. E. Lind in Melbourne and by Mr. R. W. Sheppard in Adelaide. In both Adelaide and Melbourne the highest original dilution of serum showing a "+++" or "++++" reaction was taken as the end point.

Rickettsia burneti Agglutinations.

Through the courtesy of Dr. E. H. Derrick, of the Laboratory of Pathology and Microbiology, Queensland, a number of agglutinations for *Rickettsia burneti* were performed on specimens of serum taken during convalescence. The antigen was an emulsion, prepared by several centrifugations and resuspensions of *Rhipicephalus sanguineus* ticks heavily infected with *Rickettsia burneti*. Serum dilutions were 1: 10 and 1: 30.

Histoplasmin and Coccidioidin.

Dr. J. C. Petersen, of Vanderbilt University, Untied States of America, and Dr. C. E. Smith, of Stanford University, United States of America, kindly supplied me with histoplasmin and coccidioidin. Skin tests were performed with 1: 100 dilutions of each, and the result was read for convenience at twenty-four and forty-eight hours. When the reaction to either appeared doubtful, the test was repeated with 1: 10 dilution and control, and the result was read at the same intervals.

Hirst Tests.

Through the courtesy of Professor F. M. Burnet, Hirst tests were performed at the Walter and Eliza Hall Institute in November, 1947, on suitable sera collected from patients during the study. Sera were tested against standard influenza A and B strains MMF and LEE, and also against recently isolated strains CAM (A) (9) and MIL-B (B) (10).

Subsequent Examination of Patients.

Patients were examined at intervals after recovery to ascertain the duration of convalescence, the presence of late complications, and the time taken for resolution of the pulmonary changes. Such errors in diagnosis as pulmonary tuberculosis were excluded, skin tests were performed, and further specimens of serum were obtained.

Results.

Selection of Cases.

These 14 cases were selected on the basis of clinical and laboratory criteria as representative examples of primary atypical pneumonia, ætiology unknown. The criteria used for selection permitted the inclusion of only those cases in which each of the following findings was present: (i) an acute febrile respiratory illness, conforming to the syndrome described aboue, and occurring in an otherwise healthy person; (ii) pulmonary change demonstrable by X-ray examination; (iii) adequate evidence, clinical and laboratory, that the pneumonia was not due to commonly recognized pathogenic organisms—bacteria, rickettsiæ or viruses; (iv) adequate evidence from subsequent examination that the pneumonia was not a manifestation of tuberculosis.

Owing to the shortage of beds in this hospital the more severe type of illness has predominated. These cases are presented to record the occurrence of primary atypical pneumonia in South Australia.

Ten patients were female and four were male; the ages varied from sixteen to fifty-three years. The majority of these patients became ill during the period from August, 1946, to January, 1947, when there was a small epidemic of this condition in Adelaide. Such an epidemic has not occurred since, or in the two years previous to this study. The patients, with the exception of two (Cases 103 and 128) who lived in the same hostel and came from the same district as the patients in Cases 102, 105, 109 and 120, were from widely separated areas, and all had been exposed to multiple and diverse sources of infection. Nevertheless, as a group, they exhibited a remarkable degree of clinical homogeneity. No instance of primary atypical pneumonia occurred among the staff directly responsible for the care of these patients.

Clinical Features.

The clinical features of primary atypical pneumonia observed in these 14 cases were similar to those which have been described in other reports. The onset of the illness was ill defined and gradual in 10 cases. In five of these, and in one case with an abrupt onset, the commencement of the illness was associated with mild

symptoms referable to disease of the upper part of the respiratory tract.

The presenting symptoms most frequently noted were malaise, cough, feverishness and headache, usually frontal, and severe in nature. Mild rigors were experienced by four patients, and one other patient complained of chilly sensations. Anorexia, vomiting and constipation were common, and in one case watery diarrhœa and vomiting accompanied the initial stages of the illness. Pleural pain of moderate degree was noted in three patients, but was not a prominent feature of their illness; mild chest pain, either retrosternal or felt along the costal margin, was present in several instances. Cough was usually troublesome and persistent, despite sedation with morphine or heroin; it often prevented sleep, and in five cases occurred in distressing paroxysms. As the cough became productive, scanty tenacious mucoid sputum was raised, later to become purulent and of a yellow or green hue, and occasionally streaked with blood. Two patients (Cases 108 and 109) had small hæmoptyses after lung puncture.

On physical examination of the patients, the severity of the illness appeared to be related to the extent of the pneumonic process. Moderate cyanosis was noted in several cases. Scattered medium râles and slight dulness to percussion were the most frequent findings on examination of the chest; but dulness, localized bronchial breathing and crepitations suggesting frank consolidation were common. Rhonchi were noted in several cases, and in two cases a transient friction rub was evident.

Leucocyte counts on the patients' admission to hospital gave figures ranging from 6000 to 16,000 per cubic millimetre with the exception of one case mentioned below, and differential leucocyte counts showed an absolute or relative increase of the polymorphonuclear leucocytes. As the illness progressed, particularly when it was severe, the number of leucocytes tended to rise, owing mainly to an increase in the polymorphonuclear leucocytes. Counts of 21,100 per cubic millimetre in Case 107 and 18,700 per cubic millimetre in Case 121 were recorded at the height of the disease, and 22,500 per cubic millimetre in Case 108 a few days before death. Türck cells were seen during the febrile period in several cases.

In the nine cases in which sputum was examined, smear and cultural examinations revealed the usual pharyngeal organisms, including both pneumococci and Gram-negative bacilli in Cases 107 and 108, hæmolytic streptococci in Cases 110 and 124, and a coagulase-positive staphylococcus in Case 109. In Case 120 the lungs were bacteriologically sterile *post mortem*. The sputum was not examined in Cases 103 and 105. In Cases 102 and 104 the cough did not become productive until convalescence was established. Lung punctures were performed in Cases 101, 108, 109 and 128, and no pathogenic bacteria were found in smears or culture.

The temperature was mostly of the remitting type, and usually fell by lysis over several days. The pulse rate was invariably elevated, and relative bradycardia was not observed in these cases. The respirations were usually far more frequent than normal. The fever and the rapid pulse and respiration rates in most instances were a striking contrast to the apparent well-being and mental clarity of the patient.

Subsidence of temperature by lysis and slowing of pulse and respiration rates were usually preceded by a noticeable improvement in the patient's condition. Within a month of the onset all patients began to show clinical improvement and resolution of pulmonary opacities commenced. It was not possible to predict accurately either the duration or the severity of the illness in any given case. In some cases the pneumonic process continued to spread, and the patients became dangerously ill, with intense dyspnœa. In other cases a short improvement in general condition was followed by an exacerbation of the illness.

In no case did examination of the urine reveal any abnormality. The spleen was palpable in Cases 104 and 106 during the period of fever. One patient (Case 102) on admission to hospital had several small mobile lymph glands palpable in the right posterior triangle. Another (Case 105) had severe headache, an increased cellular content in the cerebro-spinal fluid, and acute hæmolytic anæmia with leucæmoid features of the myeloid series. A third patient (Case 110), who said that she had suffered from *erythema nodosum* seventeen and again fifteen years previously, developed this condition at the height of her pneumonia. A fourth patient (Case 128) developed a left external iliac and femoral vein thrombosis on the twenty-third day of illness, as clinical improvement was occurring. A fifth patient (Case 108) died on the twenty-seventh day of illness, with involvement of all lobes in the pneumonic process. A sixth patient (Case 120) died on the seventh day of illness, with extensive pneumonia and hæmorrhagic leucoencephalitis; this case is mentioned below, but has been fully reported elsewhere.[11]

Convalescence was slow. Complaints of weakness, of persistence of cough and of shortness of breath on exertion were common. X-ray evidence of delay in return of the pulmonary condition to normal was frequent. No other sequelæ have been observed.

Radiological Features.

The character of the established pulmonary lesions divided the 14 cases into two groups. In the first group, which consisted of Cases 101, 102, 103, 106, 109 and 124, the opacities tended to be compact and localized; this is in keeping with the majority of cases of primary atypical pneumonia.[1][2][4] In serial films the appearance of the opacities differed in no way from these descriptions in density, form and distribution.

In the second group, comprising the remaining eight cases, the pulmonary lesions assumed a form which has usually been recorded as of occasional incidence in epidemics of this condition. This form occurred in several cases of the condition transmitted experimentally. The appearance suggested a widely disseminated, focal form of pneumonia. The foci were small nodules two to five millimetres in diameter, having little tendency to coalesce, or coarse, short streaks of opacity forming a reticulum, with an overlay of haze. In the majority of cases in this group the appearance of miliary nodules predominated, although the proportion of network and nodules varied in different areas of the same lung. In Cases 104, 108, 110, 121 and 128 the prevalence and distribution of the nodules were suggestive of miliary disease of the lungs. In Cases 107 and 120 the infiltration was less extensive. In Case 105, however, the reticular pattern was the more pronounced. Whichever form of opacity, nodules or reticulum predominated, the bases of the lungs were most frequently affected.

In both groups associated features such as elevation of the diaphragm, mediastinal shift and atelectatic plates not infrequently indicated some degree of collapse. Resolution followed subsidence of the acute symptoms, and as it progressed, tenting of the more central portions of one diaphragm was disclosed in certain cases. As resolution became reasonably advanced (indicated in Table I by "first negative"), abnormally increased lung markings commonly remained. These persisted for varying periods, and in some cases are still present (July, 1948); they have been considered in another report[12] as suggesting residual pulmonary fibrosis.

It cannot be too strongly emphasized that the radiological appearances at any stage of primary atypical pneumonia are not distinctive of this condition alone and may mimic many other forms of pulmonary disease, not the least among which is tuberculosis.

Serological Studies.

Serum obtained from 13 patients with primary atypical pneumonia during the acute and convalescent phases of the illness was tested for certain properties—cold agglutinins and "T" agglutinins—known to be associated with this disease, and for antibodies against viruses known to be capable of inducing acute respiratory disease in man.

Serum was tested for the presence of cold agglutinins against human group O erythrocytes. It was found that

TABLE I.

Case Number.	General.										History of Present Illness.												
	Age. (Years.)	Sex.	U.R.T.I.	Date of Onset. (Day.)	Chemotherapy.[2]	Dose in Grammes.	Time.	Admitted.	Discharged.	Total Days in Hospital.	Headache.[3]	Malaise.[3]	Rigors.	Chills.	Fever.	Chest Pain.			Constipation.	Vomiting.	Diarrhœa.	Cough.[3]	
																Pleural.	Retro-sternal.	Other.					
101	34	F.	+	29.10.46 Gradual	—	—	—	6	38	33	—	+	—	—	+	—	—	—	+	+	—	++	
102	33	F.	+	28.10.46 Gradual	—	—	—	19	38	20	++	++	—	—	—	—	—	+	—	—	—	+++	
103[1]	19	F.	—	6. 9.46 Gradual	SM.	12	5–7	7	26	20	—	++	—	—	+	—	+	+	+	—	—	++	
104	17	M.	—	7.11.46 Sudden	SD.	30	1–7	7	28	22	+++	++	—	—	+	—	—	+	—	+	—	+++	
105	44	F.	+	29. 9.46 Sudden	—	—	—	11	34	24	+++	+++	—	—	+	—	—	—	—	+	—	+++	
106	37	F.	—	27. 1.47 Sudden	—	—	—	15	31	17	+++	+++	—	—	+	+	—	—	—	+	+	++	
107	46	M.	+	10.10.46 Gradual	—	—	—	3	50	48	+++	+++	—	—	+	—	—	—	—	—	—	++	
108	53	M.	+	4.11.46 Gradual	SD.	18	8–10	10	27 Died	18	+++	+++	—	—	+	—	—	—	+	—	—	+++	
109	27	F.	—	16. 8.46 Sudden	—	—	—	8	63	56	+++	+++	+	—	+	—	+	—	—	+	—	+++	
110	38	F.	—	20. 6.47 Gradual	—	—	—	15	53	39	+++	+++	++	—	+	+	—	—	+	—	—	+++	
120	37	F.	+	9.10.46 Gradual	—	—	—	5	7 Died	3	—	+++	—	—	+	—	—	—	—	—	—	++	
121	48	F.	—	16. 8.46 Gradual	SD.	10	3–5	6	86	81	++	+++	—	+	+	+	—	—	+	—	—	++	
124	16	M.	—	1.41.46 Gradual	—	—	—	7	34	28	+++	++	+	—	+	—	+	—	—	—	—	++	
128	18	F.	—	25. 7.46 Gradual	SM.	20	3–9	9	27	19	+++	+++	—	—	+	—	—	—	—	—	—	++	

[1] Case 103—patient transferred to private hospital on tenth day of illness.
[2] Chemotherapy: "SM" = sulphamerazine; "SD" = sulphadiazine.
[3] "—" absent, "+" mild, "++" moderate, "+++" severe.
[4] "B" = blood-stained sputum.
[5] Severity of illness: a clinical impression based on febrile reaction and appearance of patient at time of maximum illness; "M" = moderately severe illness; "S" illness; "D" = dangerous illness.
[6] "LLZ" = left lower zone; "LMZ" = left middle zone; "RMZ" = right middle zone; "RLZ" = right lower zone; *et cetera*.
[7] "P" = polymorphonuclear cells; "L" = lymphocytes; "M" = monocytes; "E" = eosinophile cells; "B" = basophile cells.

the serum of 10 patients developed this property, in a titre of 1: 32 or greater, in the specimens tested within a few days of their being taken in Adelaide. The titres obtained in Melbourne by a different method, after serum had been stored for some time, showed the same tendencies, although the titre was often greater.

Changes induced in human red cells by the action of mumps and influenza virus, and certain bacterial products, render such treated cells agglutinable by practically all normal human serum. The component of normal human serum responsible for the agglutination is usually referred to as "T" agglutinin. Burnet and Anderson[13] have found that a specific agglutinin for cells altered in this fashion can be produced by appropriate immunization of rabbits. As the question arose that possible "T" agglutinins in human serum were a result of auto-immunization by red

TABLE I.—*Continued.*

Observations in Hospital.

Typical Examination.							Signs.		X-Ray Lesion.				Fever.		At Time of Maximum Fever.		Leucocyte Counts.			Chemotherapy while in Hospital.
Diarrhœa.	Cough.[a]	Dulness.	Rales.	Rhonchi.	Crepitations.	Bronchial Breathing.	Friction Rub.	Sputum.[a]	Apparent.	Clear.	Distribution.[a]	First Positive.	Last Positive.	Negative.	Days.	Maximum °F.	Pulse	Respiration.	Day after Onset	Total Leucocyte Count.[a]	Differential Leucocyte Count.[a]	
—	++	+	—	—	—	—	—	+	6	25	LLZ. LMZ.	9	35	146	10	102	100	26	7 8 21	6,100 6,900 5,600	— P. 75, L. 14, M. 10, E. 1. Türck cells seen. —	15 grammes SD. 7–9. 1,160,000 units penicillin 8–14.
—	+++	+	+	—	+	—	—	—	19	36	RMZ. RLZ. LMZ.	22	29	72	10	100	108	26	21	15,900	P. 74, L. 16, M. 7, E. 1, B. 2.	2,100,00 units penicillin 22–29.
—	++	+	+	?	?	?	?	+	5	?	RMZ. RLZ.	8	8	50	12	103	120	20	8	6,700	P. 77, L. 17, M. 5, E. 1.	19 grammes SD. 7–10.
—	+++	—	+	—	+	—	—	+	1	17	LLZ. RUZ. RMZ. LUZ. LMZ. LLZ.	8	8	26	9	102	104	20	9	9,100	P. 76, L. 8, M. 7, E. 9.	11 grammes SD. 7–9. 1,700,000 units penicillin 10–17.
+	++	+	+	+	—	+	—	+	11	23	RMZ. RLZ. LLZ.	12	27	126	5	103	116	28	12 14 19 31 150 395	36,500 24,200 5,300 3,100 8,500 5,700	Hæmolytic anæmia, leuchæmoid blood picture. See text.	18 grammes SM. 11–15. 705,000 units penicillin 11–17.
—	++	—	—	—	—	—	—	—	—	—	LUZ. LMZ. LLZ.	16	17	51	2	100	110	20	18	9,800	P. 78, L. 6, M. 11, E. 5, B. 1.	Nil.
—	++	+	+	+	+	+	—	+	3	40	RMZ. RLZ. LLZ.	6	47	142	29	102	146	32	8 16 22 30 40	7,600 21,100 13,500 12,900 8,500	— P. 86, L. 12, M. 2. Türck cells seen. — — —	32 grammes SM. 3–11. 940,000 units penicillin 6–13. 3,000,000 units penicillin 15–25.
—	+++	+	+	—	+	—	—	+ B.	8	—	RUZ. RMZ. RLZ. LUZ. LMZ. LLZ.	10	25	—	18	103	126	26	10 23	11,800 22,500	P. 82, L. 13, M. 4, E. 1. P. 83, L. 6, M. 9, E. 1, B. 1. Türck cells seen.	3,280,000 units penicillin 10–27.
—	+++	+	+	—	—	+	—	+ B.	8	60	RMZ. RLZ. LLZ.	11	350	—	22	104	120	36	9 14 21 34	10,900 14,600 9,700 10,200	P. 81, L. 7, M. 7, E. 4, B. 1. P. 79, L. 6, M. 11, E. 4. Türck cells seen. — —	16 grammes SD. 8–11. 1,000,000 units penicillin 11–16.
—	+++	+	+	—	+	—	+	+	15	44	RUZ. RMZ. RLZ. LMZ. LLZ.	18	30	41	18	102	120	40	16 27 33 46	10,300 7,800 14,000 7,700	P. 79, L. 10, M. 7, E. 4. P. 81, L. 16, M. 3. P. 71, L. 27, M. 2. Türck cells seen. P. 53, L. 41, M. 5, E. 1.	30 grammes SD. 15–20. 2,500,000 units penicillin 17–25.
—	++	—	+	+	—	—	—	+	3	—	RMZ. RLZ. LMZ. LLZ.	6	6	—	3	101	102	28	6	8,500	—	12 grammes SM. 5–7. 550,000 units penicillin 5–7.
—	++	+	+	—	+	+	+	+	3	71	RUZ. RMZ. RLZ. LUZ. LMZ. LLZ.	8	60	123	19	102	132	28	6 14 18 26 34 36	8,000 7,000 18,700 14,700 9,900 9,700	P. 85, L. 8, M. 3, E. 3, B.1. — — — P. 80, L. 10, M. 8, E. 1, B. 1. —	30 grammes SD. 9–13. 4,300,000 units penicillin 6–41.
—	++	+	+	—	+	+	—	+	7	28	LLZ.	11	11	28	8	102	116	24	8	8,300	P. 71, L. 17, M. 6, E. 5, B. 1.	32 grammes SM. 7–14.
—	++	—	+	+	—	—	—	+ B.	3	22	RUZ. RMZ. RLZ. LLZ.	9	9	22	5	104	128	46	9 13 26	8,400 11,600 6,800	P. 67, L. 17, M. 14, E. 1, B. 1. P. 77, L. 17, M. 4, E. 2. Türck cells seen. —	6 grammes SD. 13. 1,000,000 units penicillin 9–18.

cells damaged by virus, bacterial or other agents, Lind and McArthur titrated a large series of sera from humans of all ages as a preliminary approach. A further series, from patients with illnesses resembling primary atypical pneumonia, was examined to compare cold agglutinin titres for normal human cells with "T" agglutinin titres, by the use of cells treated with receptor-destroying enzyme prepared from cholera vibrio filtrates. Results suggested that the "T" agglutinin was not an immune antibody. It failed to pass the placenta, but normally developed during the first year of life, and after the age of three years the titres were not significantly different from those of adults. There was nosignificant alteration in the "T" agglutinin titre of serum from patients suffering from mumps, influenza, psittacosis and infective hepatitis. Serum from patients with primary atypical pneumonia with cold agglutinins was significantly higher in "T" agglutinins than normal serum. "T" agglutinins can be differentiated by adsorption from cold agglutinins, and it has been suggested that both reactions are "accidental" manifestations of some abnormal reactive globulin of unknown significance. The highest titres observed by Lind and McArthur in normal serum were 1:320. Of these cases presented, specimens of serum from 12 patients were titrated for "T" agglutinins, and eight showed titres of 1: 600 or greater. The evidence suggested that the titre of "T" agglutinins rose earlier and fell later than that of the cold agglutinins.

Tests for the presence of antibodies against *Rickettsia burneti* were carried out in 13 of these cases and in an additional 29 cases of suggestive acute pulmonary disease, at an appropriate interval of convalescence. No evidence of infection by this agent was disclosed in either group.

Psittacosis complement fixation tests were employed to exclude infections with the psittacosis group of viruses. Positive complement fixation by convalescent serum from patients recovered from primary atypical pneumonia has been reported, by the use of a variety of apparently unrelated antigens, from normal and infected organs of different species.[14] It is stated[15] that titres of serum from patients convalescent from psittacosis almost always rise to 1: 16 and are usually well above this figure when allantoic fluid antigens are used, while the titres of serum of patients convalescent from primary atypical pneumonia seldom exceed 1: 8 and are more often 1: 4. This was the experience in this series of cases, and in none of the cases presented was the complement fixation titre considered indicative of infection with the psittacosis group of viruses.

Paired specimens of acute and convalescent phase serum from four patients (Cases 105, 106, 110 and 124) and from 16 other patients with acute pulmonary disease admitted to the hospital during the period of this study were titrated for content of antibody against influenza viruses A and B. Although in cases of "bacterial" pneumonia it is difficult to obtain a sufficiently early specimen of serum, as an acute infection of the upper part of the respiratory tract so often precedes the pneumonia by a week or more, no evidence suggesting infection with the influenza viruses was obtained.

Agglutination tests with convalescent serum from these patients, with the exception of those in Cases 108, 110 and 120, in which the agglutination tests were not performed, gave negative results against *Salmonella typhi* "H", *Salmonella typhi* "O", *Salmonella paratyphi* A "H", *Salmonella paratyphi* B "O", *Salmonella paratyphi* B specific "H", *Salmonella choleræ suis* var. Kunzendorf "H", and *Brucella abortus*; negative results were also obtained with Proteus OX19, with the exception of agglutination to a titre of 1: 20 in Cases 101, 106 and 124, of 1: 40 in Case 103, and of 1: 80 in Case 109. These titres were not considered significant in an area in which murine typhus is endemic.

Subsequent Examination.

Patients rarely were fit for discharge from hospital earlier than four weeks after the onset of the disease. Convalescence was protracted, a further four to twelve weeks elapsing before they resumed their accustomed duties; the longer period of convalescence was usually associated with the more severe illness. The chief complaints were of physical weakness, persisting mild cough, and shortness of breath on exertion. Patients were observed until they had regained their usual health.

In six cases the Mantoux test with a dilution of 1: 1000 produced a negative response; in four cases the response

TABLE II.

Number of Case.	Onset.	Admission to Hospital.	Discharge.	Serum Obtained.	Cold Agglutination.		Psittacosis Complement Fixation.		"T" Agglutination.	Hirst Titres.				Response to Mantoux Test (1 : 1000 Dilution).
										A.		B.		
					Adelaide.	Melbourne.	Adelaide.	Melbourne.		MMF.	CAM.	LEE.	MIL-B.	
101	29.10.46	3.11.46	5.12.46	6.11.46	1 : 32									Negative 21.3.47 and 5.11.47
				14.11.46	1 : 8	1 : 30		Nil	1 : 240					
				21. 3.47	Nil	<1 : 5		1 : 10	1 : 160					
102	28.10.46	16.11.46	5.12.46	27.11.46	1 : 64	1 : 30		Nil	1 : 120					Negative 20. 3.47 and 27.10.47
				29.11.46	1 : 4									
				20. 3.47	Nil	1 : 20		Nil	1 : 40					
103	6. 9.46	12. 9.46	31. 9.46	13. 9.46	Nil									Positive for years
				1.11.46	1 : 32	1 : 40		Nil	1 : 600					
				20. 3.47	Nil	1 : 80		<1 : 5	1 : 1280					
				2.10.47				Nil						
104	7.11.46	13.11.46	4.12.46	27.11.46	1 : 128	>1 : 320		Nil	1 : 800					Positive 21. 3.47
				21. 3.47	Nil	1 : 15		Nil	1 : 120					
105	29. 9.46	9.10.46	1.11.46	17.10.46	1 : 256	1 : 80		Nil	1 : 320	16	<10	64	15	Negative 27.10.47
				29.10.46	1 : 16									
				8.11.46	1 : 32	1 : 5		Nil	1 : 320	16	<10	64	15	
				25. 2.47	Nil	1 : 8		Nil	1 : 20					
106	27. 1.47	10. 2.47	26. 2.47	17. 2.47	1 : 256	1 : 500	Nil		1 : 2560	120	<10	112	64	Negative 22.10.47
				22. 2.47	1 : 128	1 : 500			1 : 2560	120	<10	112	64	
				12. 3.47	1 : 2	1 : 200	Nil	Nil	1 : 1280					
107	10.10.46	12.10.46	28.11.46	25.10.46	1 : 128	1 : 640		Nil	>1 : 1280					Positive 19.11.47
				1.11.46	Nil		1 : 12							
				28. 2.47		1 : 20		<1 : 5	1 : 3200					
108	4.11.46	13.11.46	30.11.46 Died	26.11.46	1 : 128	>1 : 320		Nil	1 : 1560					
109	16. 8.46	23. 8.46	17.10.46	30. 8.46	1 : 64									Positive 1937
				17.10.46	Nil									
				18. 3.47			Nil							
110	20. 6.47	4. 7.47	11. 8.47	7. 7.47	1 : 256			Nil	1 : 640	30	<10	140	40	Negative 4. 8.47 and 27.10.47
				22. 7.47	1 : 160			Nil	1 : 2350	56	<10	240	80	
				4. 8.47	1 : 32			Nil	1 : 1280	56	<10	240	80	
121	16. 8.46	21. 8.46	9.11.46	14. 9.46	Nil									Negative 3.10.46 Positive 13. 3.47
				26. 9.46	Nil	<1 : 5	Nil		1 : 90					
				13. 3.47										
124	1.11.46	7.11.46	4.12.46	9.11.46	1 : 2	<1 : 5		Nil	1 : 1600	80	<10	>320	256	Negative 1943
				21.11.46	1 : 16	1 : 80		Nil	1 : 1600	80	<10	>320	256	Negative 17. 3.47
				17. 3.47		1 : 10	Nil	Nil	1 : 1900	80	<10	>320	256	Positive 20.11.47
128	25. 7.46	2. 8.46	20. 8.46	6. 8.46			Nil							
				20.12.46	Nil	<1 : 20	<1 : 6	Nil	1 : 640					Negative 11. 3.47 and 12.11.47
				11. 3.47	Nil	1 : 20	Nil	1 : 10	1 : 275					

was positive; in the two fatal cases (108 and 120) it was not performed. In Case 121 the Mantoux test with a dilution of 1: 1000, which had produced a negative response on October 3, 1946, in early convalescence, produced a positive response on March 13, 1947. Because of the long period in hospital the possibilities of infection in the ward or of a period of anergy to tuberculin following a severe illness must be considered. The pneumonia was probably not a manifestation of tuberculosis, as examination of eight smears failed to reveal acid-fast bacilli in the sputum. Clinical recovery occurred and the pulmonary opacities had largely resolved within sixty days of onset. In Case 124 the Mantoux response to a dilution of 1: 1000 was negative in 1943 and on March 17, 1947 (four and a half months after onset of the pneumonia), but was found to be positive on November 20, 1947; this indicated infection since March 17, 1947.

Histoplasmin and coccidioidin skin tests were performed in all cases, from three to thirteen months after the onset of the illness, with the exception of Cases 108 and 120, which were fatal. All tests produced negative results.

Differential Diagnosis.

The acute onset, the severe pleural pain, the rusty sputum, the early leucocytosis and the response to chemotherapy often serve to differentiate the coccal pneumonias; but cases of pneumococcal pneumonia closely resembling primary atypical pneumonia and resisting chemotherapy do occur.[16] In the aged or predisposed the bacterial pneumonias often present an unusual course and fail to respond to chemotherapy. Purulent bronchitis and staphylococcal tracheo-bronchitis have frequently an acute onset and produce abundant expectoration of sputum. Pulmonary signs frequently accompany septicæmia caused by bacteria. Also sustained pyrexia may accompany the administration of sulphonamides in certain persons. Pulmonary embolism, infarction and thrombosis, which may present divers appearances, are relatively uncommon in the healthy young adult, but become increasingly frequent when bed rest is necessary, particularly in the presence of cardiovascular disease. Mildly infected atelectasis of lobules, segments or lobes may arise during the course of various acute and chronic catarrhal affections of the upper part of the respiratory tract, the result of obstruction of bronchi or bronchioles by infected mucus of nasal or bronchial origin.

Primary atypical pneumonia of restricted extent may clinically and radiologically resemble pulmonary tuberculosis, whilst the diffuse type may simulate miliary tuberculosis. Pleural effusion, coexisting with the pneumonia, when of considerable extent and tending to persist, is more safely regarded as a manifestation of tuberculosis.

Pneumonia due to fungi and yeasts, particularly primary coccidioidal pneumonia,[17] may closely resemble primary atypical pneumonia; the diagnosis rests upon the isolation of the fungus from the sputum or a positive response to the coccidioidin skin test.

Acute pulmonary disease may follow the inhalation of irritant gases, vapours or other matter, and may be differentiated by the history of recent exposure to such noxious material.

"Tropical lung" is characterized by high eosinophilia, pyrexia, disseminated pulmonary opacities and response to arsenic. Transient pulmonary infiltrations associated with eosinophilia characterize Löffler's syndrome. Other "allergic" pulmonary infiltrations occur in *erythema multiforme exudativum*,[18] and have been reported during therapy with sulphonamide.[19]

Empyema may be confusing, as also may spreading suppurative pneumonitis and lung abscess. Atelectasis, bronchiectasis, hydatid disease, neoplasm or metastases may simulate primary atypical pneumonia.

Diseases caused by the viruses of influenza, by the psittacosis group of viruses, or by *Rickettsia burneti* (Q fever), may closely resemble primary atypical pneumonia, and differentiation may be possible only after isolation of the infective agent or demonstration of increase of antibody to the agent in question. Pneumonia may be present in the acute exanthemata, particularly morbilli and varicella. Pertussis may be mimicked by primary atypical pneumonia with paroxysmal cough.

It is worthy of note that pulmonary lesions have been described accompanying infectious mononucleosis[20] and lymphocytic choriomeningitis,[21] and that a pulmonary form of leptospirosis does occur.[22]

Treatment.

Treatment was directed to relief of symptoms. Alleviation of the headache was obtained often by aspirin, but in one case lumbar puncture was performed with little relief. Cough resisted most measures, but some success was obtained by the use of heroin, and in some instances sweetened drinks also helped to allay pharyngeal irritation. Morphine was frequently used to diminish the rapid, shallow respirations, to depress the cough reflex, and to promote deeper respiration. Oxygen was administered intranasally in Cases 107, 108, 110 and 121, with beneficial effect and lessening of the dyspnœa.

Although sulphonamides and penicillin were frequently employed in adequate dosage, in no instance was any response observed. In some instances, particularly among the older patients, the appearance of copious purulent sputum and exacerbation of the cough suggested the advent of secondary infection, despite the administration of penicillin and sulphonamide at the time. While this gave the impression of prolonging the illness, no material alteration in the X-ray appearance of the lungs was detected.

Discussion.

Pneumonia may be caused by a wide variety of agents, some inanimate like the noxious gases or mineral oils; but most cases of pneumonia are due to microorganisms. Since the urgent need for specific agents to combat pneumonia caused by bacteria has been so well met in the majority of cases by the sulphonamides and penicillin, interest has turned to the residue of cases not affected by chemotherapy. Unusual forms of tuberculosis, pneumonia due to certain Gram-negative organisms such as Friedländer's bacillus, or fungous disease, such as coccidioidomycosis in certain parts of the world, account for a proportion of these cases. It must be stressed, in this group of "resistant" pneumonias, that the course of bacterial pneumonia, ordinarily responsive to chemotherapy, may be greatly altered by local or general disease, which lowers the capacity of the patient to deal adequately with the infection. Pulmonary embolism and infarction, septic or aseptic, are by no means uncommon conditions and are protean in the variety of their appearance and clinical course.

In the remainder of this group of "resistant" cases no causal organism was found by ordinary bacteriological methods, and these cases have been grouped together as atypical or viral pneumonia. Closer investigation of this group has shown that it too is heterogeneous. Some of the causative viruses are known; others remain to be isolated.

In an American series some 10% of such cases proved to be due to viruses of the psittacosis group,[23] and epidemics of atypical pneumonia in Italy and America were proved to be due to *Rickettsia burneti*.[24] Pneumonia has accompanied infection with the viruses of influenza, lymphocytic choriomeningitis and infectious mononucleosis. The majority of the remaining cases, occurring in sporadic but frequently epidemic form, had a number of unusual features in common—notably the presence of cold agglutinins in the convalescent serum in a proportion of cases. The name "primary atypical pneumonia, ætiology unknown" has been agreed to for this syndrome. The fact that this condition should occur in epidemics of itself suggests a common ætiological agent. Further, the Commission on Acute Respiratory Diseases was able to transmit such infections to human volunteers by the inhalation of filtrates of infective material, thus establishing a virus or viruses as the causative agent. The commission found the incubation period to be approximately one week in subjects receiving untreated inoculum, and two weeks in those inoculated with the filtrate. More recent transmission experiments by the commission suggest that a disease resembling the common cold, with predominantly nasal

symptoms and incubation period of two days, and another disease with predominantly pharyngeal symptoms and incubation period of six days, very like the epidemic pharyngitis of new recruits, are immunologically distinct entities, and previous infection with these diseases did not prevent infection with primary atypical pneumonia.[25]

In the investigation carried out at the Royal Adelaide Hospital over a period of fourteen months it was found that 15% of the cases of pneumonia studied conformed to the descriptions of naturally acquired or transmitted primary atypical pneumonia, ætiology unknown. Adequate investigations were carried out in only fourteen of these cases, which have been presented in this paper. Although serological investigations for influenza and Q fever were carried out in these and in other cases of acute pulmonary infection, no positive results were obtained. No serological evidence of psittacosis was detected in the cases presented in this paper, although psittacosis is known to occur in South Australia,[26] and I have seen a number of patients with this condition during this investigation. Q fever has recently been recognized in this State.[27] The significance of a rise in titre of the "T" agglutinins is by no means established in relation to primary atypical pneumonia and other diseases, as the number of cases so far studied has been small. One patient suffering from pneumonia due to infection by Friedländer's bacillus had an abnormally high titre of "T" agglutinins.

No previous investigation had been made upon the occurrence of coccidioidomycosis or histoplasmosis in South Australia. By the use of coccidioidin and histoplasmin skin tests were carried out in the cases described here, upon a number of adults, some healthy and some with chronic lung diseases, and mostly upon children. One hundred and sixty coccidioidin and 100 histoplasmin skin tests have been carried out; all gave negative results.

The exclusion of recognized causal organisms of other forms of pneumonia, the resemblance of the clinical picture and the radiological features, and the frequent occurrence of cold agglutinins, indicate that the majority of cases here described were of primary atypical pneumonia, ætiology unknown. The pulmonary lesions in the two fatal cases presented many of the features of acute interstitial pneumonitis recorded by Golden,[28] who has described the pathology of this condition from selected autopsy material, in cases in which the clinical diagnoses were considered satisfactory.

Of clinical interest is the occurrence of leucoencephalitis in one case, and in another case that of hæmolytic anæmia with leucæmoid features in the myeloid series, also with changes in the cerebro-spinal fluid. Encephalitis has been noted accompanying primary atypical pneumonia clinically and at autopsy by many observers, and also in association with other forms of pneumonia, and has been produced experimentally. However, this has been fully discussed elsewhere by French and Fowler in the presentation of this case.[11] A blood picture resembling that found in lymphatic leuchæmia accompanying a case of primary atypical pneumonia, ætiology unknown, has been reported previously,[29] but no instance of accompanying hæmolytic anæmia with leucæmoid picture of the myeloid series.

Nothing new in the clinical or serological picture has appeared during these studies. The X rays reveal a feature which is mentioned incidentally by other writers, but in this series of cases appeared in high proportion. That is a widely disseminated focal type of opacity, with scattered small nodules and coarse streaks forming a reticulum, occurring over a considerable area of the lungs, sometimes aggregated into small clumps of moderate density, often granular in appearance, not occurring in dense fluffy shadows as found in lobar pneumonia, and rather miliary in distribution.

Before this discussion is concluded some speculation upon the pathogenesis of primary atypical pneumonia, ætiology unknown, may be permissible. The designation indicates that infection is regarded as being due to inhalation of the causative agent or agents, and to growth and spread within the lung. This undoubtedly occurs in influenza, but several indications suggest that it may not occur in primary atypical pneumonia. The important points are as follows: (i) the minimal incubation period of seven days is much longer than one would expect in purely local pulmonary disease (contrast that of influenza, two days); (ii) the widespread, often symmetrical, distribution of the pulmonary lesions is similar to that seen in primary measles pneumonia and in miliary tuberculosis, which are due to dissemination of the causative organism by the blood-stream; (iii) in primary atypical pneumonia enlargement of the spleen sometimes occurs, and in an acute illness this of itself suggests hæmatogenous spread. Leucoencephalitis may accompany or follow other forms of pneumonia, and it has been suggested that cold agglutination may be responsible for hæmolytic anæmia. However, the occurrence of leucoencephalitis in Case 120, and in Case 105 of hæmolytic anæmia with myeloid primitive cells and symptoms of encephalitis with an increased cellular content of the cerebro-spinal fluid, is suggestive of a general disease. The occurrence of cold agglutinins and "T" agglutinins, like the heterophile agglutinins in mononucleosis, indicates the presence of some widespread interference with the globulin-producing mechanism.

These features suggest that primary atypical pneumonia, ætiology unknown, may be a general systemic disorder which is almost invariably localized in the lungs. Other forms of virus pneumonia, Q fever and psittacosis are known to have a phase in which the organisms occur in the blood. It may be, therefore, that the sequence of events in this condition consists of the occurrence of a primary localized focus of infection in the respiratory tract, which may either spread directly along the mucous membranes to produce a rather extensive local condition or be followed by invasion of the blood-stream via the regional lymph nodes. Most frequently, perhaps, both methods of spread occur. The widespread infection of the lungs may directly follow this period of viræmia; or possibly there may be, as Fenner[30] (1948) has postulated for the acute exanthemata, a period of multiplication in the spleen and elsewhere in the cells of the reticulo-endothelial system before sufficient virus is freed into the blood-stream to cause the widely scattered foci of infection in the lungs.

Summary.

1. The clinical features of 14 cases of primary atypical pneumonia, ætiology unknown, treated at the Royal Adelaide Hospital between June 1, 1946, and October 1, 1947, have been described.

2. The symptomatology, physical findings, radiographic pattern and clinical course of disease in these cases conform in the main to descriptions of the disease transmitted to human volunteers and of the naturally acquired infection.

3. Serological studies are presented in detail, including cold agglutinin, "T" agglutinin and psittacosis complement fixation titres.

4. Autopsy findings are reported in two fatal cases of this condition, one with accompanying leucoencephalitis.

5. One case with an accompanying acute hæmolytic anæmia and leuchæmoid features of the myeloid leucocytes is presented.

6. No serological evidence of the occurrence of Q fever was obtained.

7. A number of skin sensitivity tests to coccidioidin and histoplasmin have been performed. All gave negative results.

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Appendix.

Case Summaries.

Three cases are presented in detail. The first case is presented because of some points of unusual interest, and the last two cases, which terminated fatally, are included with relevant autopsy findings. Clinical data of these cases are presented in Table I, serological data in Table II.

CASE 105.—A *multipara*, aged forty-four years, was admitted to hospital on October 9, 1946. Ten days previously she had noticed a tight feeling in her throat, soon followed by a severe headache and pain behind the eyes, an unproductive cough and a feeling of tightness in the chest. These symptoms caused her to stay in bed, where she had remained, feeling feverish, and perspiring freely at times. The cough increased in severity and became productive of a small amount of grey sputum, and she had no appetite during this time. On the two days prior to her admission to hospital, cough had occurred in paroxysms so severe that she felt as though she was suffocating; she was not able to sleep because of nocturnal coughing, and had vomited frequently. There was no history of previous pneumonia, but as a child she had had severe whooping-cough, and had coughed up blood during a paroxysm.

Physical examination revealed the patient to be a middle-aged woman, slightly obese, with skin and conjunctivæ tinged with yellow, who was breathing quietly and appeared moderately ill. The temperature was 99·6° F., the pulse rate was 112 per minute and the respirations numbered 24 per minute. The ocular fundi were normal. The tongue was dry and coated, the fauces were inflamed with numerous small patches of white exudate, but no cervical lymph glands were palpable. The heart was not enlarged, the sounds were normal, and the blood pressure was 110 millimetres of mercury (systolic) and 50 millimetres (diastolic). The percussion note was slightly impaired, and breath sounds were of diminished intensity at the base of the right lung; râles and rhonchi were heard over both lungs. There were no neurological signs and no alteration of the reflexes.

Treatment was commenced with both sulphamerazine, 18 grammes being given in the next four days, and penicillin, a total of 705,000 units being given intramuscularly from October 9 to 15, 1946. "Ascotin" was given for relief of headache, heroin to control the cough, and iron by mouth for the anæmia. On October 11 the patient was feeling very miserable with severe headache and aching in the limbs; her evening temperature was 103° F., her pulse rate was 116 per minute, and her respirations numbered 28 per minute. No lymph glands were palpable, nor was the spleen or liver palpable. On October 13 lumbar puncture was performed; the pressure of cerebro-spinal fluid was normal, and there was a normal response to jugular compression. There was little relief of the headache. On October 15 the elevated temperature had subsided, the patient felt much better, the cough had become less, and the headache had abated considerably. A few râles were audible at the extreme bases of both lungs posteriorly. On October 26 headache was still present, and there was no discoloration of skin or conjunctivæ, very little cough and no sputum. The patient was discharged from hospital on November 1 with mild headache, but otherwise well. At no time had there been any neurological signs. No blood transfusion was given.

Blood counts gave the following information. On October 10 the hæmoglobin value was 5·9 grammes *per centum*, the relative volume of packed cells was 19%, reticulocytes numbered 8%, 11% of the erythrocytes were nucleated and platelets were plentiful; the leucocytes numbered 56,500 per cubic millimetre, 56% being polymorphonuclear cells, 11% metamyelocytes, 10% myelocytes, 1% myeloblasts, 8% lymphocytes, 1% eosinophile cells and 2% basophile cells. Macrocytes were present, and examination of the erythrocytes revealed pronounced anisocytosis and polychromasia. The average diameter of the erythrocytes was slightly increased. The plasma was hæmolysed, but had a yellow tinge. On October 12 the leucocytes numbered 24,200 per cubic millimetre, 70% being polymorphonuclear cells, 5% metamyelocytes, 5% myelocytes and 4% lymphocytes; 16% of the erythrocytes were nucleated. Myeloblasts and Türck cells were present. Examination of the erythrocytes revealed a slight increase in average diameter, anisocytosis and many macrocytes. On October 17 the hæmoglobin value was 8·0 grammes *per centum*; 15% of the erythrocytes were reticulocytes, 2% were nucleated and platelets were plentiful. The leucocytes numbered 5,300 per cubic millimetre, 71% being polymorphonuclear cells, 19% lymphocytes, 5% monocytes and 3% eosinophile cells. Macrocytes were present, and examination of the erythrocytes revealed a slight increase in average diameter, pronounced anisocytosis and polychromasia. On October 29 the hæmoglobin value was

12·8 grammes *per centum*; 4% of the erythrocytes were reticulocytes and platelets were plentiful; the leucocytes numbered 3100 per cubic millimetre, 56% being polymorphonuclear cells, 31% lymphocytes, 8% monocytes, 4% eosinophile cells and 1% basophile cells. Anisocytosis was present, and the average diameter of the erythrocytes was normal. An erythrocyte fragility test performed on October 24 gave results within normal limits.

Cold agglutinin titres were 1:256 on October 17, 1:16 on October 29, 1:32 on November 8 and nil on February 25, 1947. The cerebro-spinal fluid on October 13 was clear and colourless, it contained no excess of globulin, and the protein content was 30 milligrammes per 100 millilitres. A cell count revealed 30 erythrocytes and 40 leucocytes per cubic millimetre, of which 80% were lymphocytes and 20% polymorphonuclear cells. The fluid was bacteriologically sterile. The sputum was not examined.

X-ray examination of the chest on October 10 revealed extensive involvement of the right lung and of the base of the left lung. On October 25 most of the opacity had resolved, but there remained a linear opaque area suggesting atelectasis in the lower part of the right lung.

On February 25, 1947, when next seen, the patient was well, had not regained her former vigour, and still had a slight cough. A blood count gave the following information: the hæmoglobin value was 13·4 grammes *per centum*, and 0·5% of the erythrocytes were reticulocytes; the leucocytes numbered 8500 per cubic millimetre, 44% being polymorphonuclear cells, 44% lymphocytes, 8% monocytes, 2% eosinophile cells and 2% basophile cells. Platelets were plentiful. X-ray examination revealed a few scattered, ill-defined, residual opacities at the base of the left lung. The patient said that whilst she was in hospital her husband had been ill for one week with fever, headache and cough, but had continued work.

On October 20, 1947, the patient was well, but three weeks previously she had had a further attack of "pneumonia", with headache less severe than previously and cough of paroxysmal nature. However, she had been treated at home by her physician with sulphonamide tablets, and the illness had cleared up within a week. The radiological appearance of her lungs had altered little since the previous examination, and a blood count gave the following information: the hæmoglobin value was 13·9 grammes *per centum*, platelets were plentiful, and 2% of the erythrocytes were reticulocytes; the leucocytes numbered 5800 per cubic millimetre, 58% being polymorphonuclear cells, 30% lymphocytes, 8% monocytes, 1% eosinophile cells and 1% basophile cells. The differential leucocyte counts were repeated on October 29, 1947, and the figures had not altered materially. The ocular fundi were normal, there were no palpable lymph glands, and the spleen and liver were not palpable.

Investigation of the cerebro-spinal fluid obtained on October 13, 1946, was carried out by Mr. E. L. French, who kindly supplied the following particulars. Cerebro-spinal fluid was injected into two rabbits, two guinea-pigs, and six mice intracerebrally. The temperatures of the rabbits were taken daily, and over a period of fourteen days both animals had normal temperatures. They were kept under observation for a further period of three months and then destroyed. The mice and guinea-pigs also appeared normal over this period of observation. Cerebro-spinal fluid was inoculated into developing chick embryos, both intraamniotically and onto the chorio-allantoic membrane. The inoculated eggs were passaged six times and no lesions were detected at any stage. Amniotic fluid was tested with known susceptible fowl cells for hæmagglutinin due to influenza virus. The fluid gave negative results on each occasion.

The investigations failed to reveal the presence of a virus in the cerebro-spinal fluid.

CASE 120.—A female patient, aged thirty-seven years, was admitted to hospital on October 13, 1946. For five days she had had a "cold on the chest", with cough productive of yellow mucoid sputum, and some difficulty in breathing. Two days prior to her admission to hospital she was examined by her own physician, who noted that the symptoms were those of an acute upper respiratory tract infection, and that numerous coarse râles were present throughout the lungs. Sleep had been poor because of cough and shortness of breath. The following day weakness of the left arm appeared. Appendicectomy had been performed at the age of seventeen years, and an ovarian cyst removed at the age of twenty-eight years.

On examination of the patient shortly after her admission to hospital, the temperature was 100° F., the pulse rate was 104 per minute, and the respirations numbered 40 per minute; the patient appeared distressed, but was coherent. The pupils were not abnormal; the tongue deviated to the left on protrusion and was coated with a white fur. Left facial palsy of upper motor neuron type was present. The heart seemed normal, and the blood pressure was 140 millimetres of mercury (systolic) and 80 millimetres (diastolic). The respiratory excursion of the chest was poor, and numerous râles and rhonchi were heard over both lungs. Flaccid paralysis of the left arm was present, with anæsthesia to touch and pressure. The tendon reflexes were slightly increased in the left arm. There was weakness of the left leg, with anæsthesia to touch and pressure, exaggerated reflexes, and an extensor plantar reflex; the plantar reflex of the right foot was flexor in type.

On the evening of October 13 the patient became comatose, but stirred when painful stimuli were applied. The breathing was stertorous, and her pupils were small, circular and equal, and reacted to light. The following day the patient could be roused for short periods, but she died on October 15. Since her admission to hospital 12 grammes of sulphamerazine had been given by mouth, and 550,000 units of penicillin had been given intramuscularly.

The laboratory findings were as follows. A blood examination on October 14 revealed that the hæmoglobin value was 14·0 grammes *per centum*, that 2% of the erythrocytes were reticulocytes, and that the leucocytes numbered 8500 per cubic millimetre. Lumbar puncture on October 13 produced fluid under a pressure of 120 millimetres; there was a normal response to jugular compression. The cerebro-spinal fluid was slightly cloudy, and contained five erythrocytes and 220 leucocytes per cubic millimetre, 95% being polymorphonuclear cells and 5% lymphocytes. The fluid contained an excess of globulin, and 125 milligrammes of protein and 100 milligrammes of sugar, per 100 millilitres. On October 14 the cerebro-spinal fluid was turbid and contained five erythrocytes and 1300 leucocytes (95% polymorphonuclear cells, 5% lymphocytes) per cubic millimetre. The chloride content was 0·70 milligramme per 100 millilitres. No bacteria were seen in direct smears of the fluid.

An X-ray film of the chest on October 14 (of poor quality) revealed scattered nodular opacities in the lower half of both lungs.

Post-mortem examination twenty-three hours after death revealed that the diaphragm was elevated to the fourth anterior intercostal space on both sides; the lungs appeared distended with air, and there were slight pleural adhesions at the base of the left lung, and at the apex and base of the right lung. The tracheal mucous membrane was slightly congested, the bronchial and tracheal lymph nodes were large and œdematous, and the bronchi were congested and contained mucopus. In the lower lobes of both lungs were extensive purple, sunken, airless patches, and the intervening lung tissue was pale and œdematous. On incision of the lungs, these areas were studded with pale, firm "tubercles"—approximately three-quarters of the lower lobe of the left lung and one-third of the lower lobe of the right lung were affected. There was a small calcified speck at the apex of the left lung. In the brain, the meninges appeared normal, although the pial vessels were congested and the convolutions flattened. The right cerebral hemisphere was larger than the left, and about the middle of its lateral surface was intensely specked with hæmorrhagic puncta and felt softened. Specking was not obvious over the left hemisphere, but the *corpus callosum* appeared soft.

After fixation, these hæmorrhagic puncta, historically ball hæmorrhages or necrosis round a small vessel surrounded on the outside by hæmorrhage, involved chiefly the white matter. They occurred mainly in the right *centrum semi-ovale*, and were fewer and more restricted in extent in the left *centrum semi-ovale*, and the *corpus callosum* was affected. The lesions could be traced through the internal capsule into the *basis pedunculi;* again the most obvious damage was on the right side. Similar changes were noted beneath the floor of the aqueduct in the mid-line, in the superior and middle peduncles and the cerebellum itself, and in the lateral walls, floor and roof of the fourth ventricle. The closed part of the medulla was not affected. Both optic tracts were affected.

Microscopic examination of the lungs in the darker areas revealed congestion of the smaller vessels of the alveolar capillaries and particularly of the submucosal and extramuscular bronchiolar vessels. The septa and the perivascular and peribronchiolar connective tissue were œdematous and densely infiltrated with lymphocytes. The bronchiolar lumina were filled with inflammatory casts consisting mainly of polymorphonuclear cells; the epithelium of the bronchioles was partly desquamated and replaced by granulation tissue—monocytes, macrophages, fibroblasts and a few lymphocytes. Examination of the bronchiolar walls revealed dilated capillaries and lymphocytic infiltration. In some bronchioles, granulation tissue extended into the debris within the lumen; the bronchiolar plug was organized, and this organization

extended into terminal bronchioles and their alveoli, and the alveoli were collapsed. In the granulations were occasional giant cells with several nuclei imitating Langhans' type. Peribronchiolar alveoli were at times implicated, and in places organization of their contents was proceeding. Most of the remaining alveoli contained macrophages, with a little serous fluid here and there, and interstitial changes in the alveolar walls were infrequent. Masson's trichrome stain revealed the presence of recently formed collagen in the organizing exudate within the lumina of bronchioles, extending into the alveoli, and in some peribronchial alveoli. In several places there was present a recent polymorphonuclear exudate filling groups of alveoli, sharply demarcated from neighbouring areas of organization, which presented a striking contrast. Material taken from the lungs *post mortem* for smear and cultural examination was bacteriologically sterile, and attempts to isolate a virus met with no success.

CASE 108.—A male patient, aged fifty-three years, was admitted to hospital on November 13, 1946. Nine days previously he had noticed malaise and vague abdominal discomfort, and had gone to bed. He was no better two days later, when he had a sore throat and slight cough. His condition improved over the next few days, but on November 10 he felt very feverish, and his cough was worse and had become productive of white phlegm streaked with brown. The next day sulphadiazine treatment was commenced, and 18 grammes were given in two days without response. He was admitted to hospital complaining of an irritating paroxysmal cough, which prevented him from sleeping and caused exacerbation of a severe frontal headache. His appetite was poor, and he had been constipated for some days. Significant previous illnesses were typhoid fever at the age of twenty years and periodical severe "colds in the head" over the last five years.

On examination of the patient, he was seen to be a middle-aged man of good physique, his lips were slightly cyanosed, he was distressed by bouts of coughing, and he appeared moderately ill. His temperature was 99·5° F., his pulse rate was 88 per minute, and his respirations numbered 24 per minute. His tongue was moist and coated with a brown fur. No abnormality was detected in the heart; the blood pressure was 130 millimetres of mercury (systolic) and 70 millimetres (diastolic). Movement of the right side of the chest was impaired, and dulness to percussion was present at the apex of the right lung, with crepitations over the upper part of the right lung posteriorly and at the base of the left lung.

Heroin was given for the cough, and the intramuscular administration of penicillin was commenced. For the first three days the temperature was sustained at 102° F., and medium râles were heard over the upper part of the right lung. The cough was distressing and productive of yellow purulent sputum. There was some improvement for the next few days, although a lung puncture on November 19 was followed by a brisk hæmoptysis (one ounce). This was treated by rolling the patient on the affected side and giving morphine hypodermically. However, his temperature rose to 102° F., his pulse rate to 126 per minute, and his respiratory rate to 30 per minute on the evening of November 22. Next day râles had extended throughout the right lung and were also audible at the base of the left lung. The heart was not enlarged, but the liver edge was palpable two fingers' breadth below the right costal margin. On November 24 his condition had deteriorated, and his cough was particularly distressing.

On November 26 the patient was drowsy, but his intelligence was unimpaired; his lips were cyanosed, and the cough was productive of copious thin purulent sputum. Medium râles were heard over both lungs, the heart had become enlarged, the apical impulse was feeble, and the heart sounds were faint, with a short superficial to-and-fro squeak just inside the apex.

The intranasal administration of oxygen was commenced, but the condition remained unaltered until the patient died on November 30, despite supportive treatment, but without developing peripheral œdema; 3,280,000 units of penicillin had been given intramuscularly.

The laboratory findings were as follows. A blood count on November 13 gave the following information: the leucocytes numbered 11,800 per cubic millimetre, 82% being polymorphonuclear cells, 13% lymphocytes, 4% monocytes and 1% eosinophile cells. On November 26 the hæmoglobin value was 17·2 grammes *per centum*, 3% of the erythrocytes were reticulocytes and platelets were plentiful; the leucocytes numbered 22,500 per cubic millimetre, 83% being polymorphonuclear cells, 6% lymphocytes, 9% monocytes, 1% eosinophile cells and 1% basophile cells. Türck cells were present.

On November 14 attempted culture of the sputum produced no pathogenic bacteria, but a few organisms resembling pneumococci were present in a smear on November 15, while smears examined on November 16, 21 and 22 contained no pneumococci. Lung puncture material obtained on November 19 was sterile. Smears on November 23 contained pneumococci and on November 25 Gram-negative bacilli. Culture on November 26 produced a growth of small Gram-negative bacilli and pneumococci. Culture on November 29 produced small Gram-negative bacilli (not Hæmophilus), a coliform bacillus, a yeast, and a penicillin-sensitive pneumococcus. Blood obtained on November 29 was sterile after five days' incubation at 37° C.

Cold agglutinins were present in the serum to a titre of 1:128 on November 26.

X-ray examination on November 13 revealed opacity of the upper half of the right lung. On November 28 this opacity has resolved considerably, the cardiac shadow had increased considerably in size, but did not suggest a pericardial effusion, and ill-defined nodular opacities were scattered throughout both lungs.

Post-mortem examination, performed forty hours after death, revealed voluminous lungs which did not collapse when the chest was opened, emphysema of the anterior lung margins, and appreciable œdema and loss of elasticity posteriorly. There were no pleural adhesions. Tracheobronchial lymph glands were large and œdematous. Below the tracheal bifurcation, the bronchial mucosa was bright red in colour. On section, all lobes of the lungs had a beefy red appearance, and pus exuded from the bronchioles. The heart was larger than normal, as the right ventricle was dilated to three times its normal capacity. All valves appeared normal, the coronary orifices were patent, and the cardiac muscle was of good colour and firm texture. The liver was enlarged and congested with a nutmeg appearance. The capsules of both kidneys were adherent, and the kidneys congested. The brain at the post-mortem examination and after fixation appeared normal.

Microscopic examination of the lungs revealed congestion of the smaller vessels, of the submucosal and extramuscular capillaries of the bronchioles, and of the alveolar capillaries. The septa and the perivascular and peribronchial connective tissues were œdematous and densely infiltrated with monocytes, lymphocytes, plasma cells, phagocytes and occasional polymorphonuclear cells. Several small but sharply demarcated areas within the peribronchial tissue contrasted with the above, as they were infiltrated with polymorphonuclear cells.

In the bronchioles, the epithelium was desquamated and the lumina were filled mainly with polymorphonuclear cells and epithelial debris. In some, where the epithelium had not been completely denuded, foci of "pseudo-stratification" were seen; in others resolution had commenced with organization of the exudate in some areas, and the corresponding alveoli were collapsed. The submucosa and bronchiolar walls were infiltrated with monocytes, lymphocytes, plasma cells and occasional polymorphonuclear cells.

Alveolar changes, although frequent, were less noticeable than the bronchiolar lesions. Isolated groups of alveoli, not necessarily abutting on the peribronchiolar tissue, were seen with infiltrated and thickened walls. Some of these alveoli contained serous exudate with a few monocytes and macrophages. In other areas, the architecture was no longer distinguishable, as organization was taking place, and new collagen fibres were present.

Fat stains showed absorption of material by alveolar phagocytes and accumulation of these cells in the peribronchial tissue.

In the tracheo-bronchial lymph nodes sinus catarrh was present, in the liver portal congestion was found, and the spleen also was congested. On macroscopic examination the brain appeared normal. Unfortunately, the myocardium was not examined histologically.

Comment.

In both this and the preceding case, histological examination of the lungs revealed certain features in common. There was an acute purulent bronchiolitis with desquamation of the epithelium; occlusion of these bronchioles in varying degree appeared responsible for collapse or emphysema of the corresponding alveoli. As a great contrast, the walls of such bronchioles were infiltrated chiefly with mononuclear cells, the infiltration extending radially into the regional interstitial tissues of the lung. The alveoli were relatively free of polymorphonuclear leucocyte exudate, the findings thus differing from those obtained in lobar pneumonia and bronchopneumonia.

Although death occurred in Case 120 seven days after onset, and in Case 108 nine days after apparent spread of

the process to the remaining areas of both lungs, in both cases there was variation in the age of the bronchiolar lesions. Recent polymorphonuclear exudate in some bronchioles contrasted with the mononuclear cells and phagocytes in the lumina of neighbouring bronchioles. In some of the latter the plug of exudate was already invaded by granulation tissue. In the alveoli surrounding such bronchioles, areas of organization were to be seen. These findings suggest the possibility of later pulmonary fibrosis and incomplete resolution following this condition.

FIBRINOGEN B: A PRELIMINARY SURVEY OF THE INCIDENCE OF FIBRINOGEN B IN NORMAL AND DISEASED STATES.

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FOLLOWING the investigation by Cummine and Lyons[1] of the clinical significance of fibrinogen B in post-operative thrombosis, a survey was commenced to determine the incidence of fibrinogen B in other conditions. Although this survey is incomplete at the moment, some interesting observations have been made.

TABLE I.

Subjects.	Total.	Number with Fibrinogen B.	Percentage Occurrence of Fibrinogen B.
Normal subjects	2,240	9	0·4
Medical patients	294	154	52
Surgical patients ..	286	162	57
Pregnant women	200	30	15

TABLE II.

Investigation.	Total.	Number with Fibrinogen B.	Percentage Occurrence of Fibrinogen B.
Normal subjects	240	4	1·7
Stored citrated blood ..	2,000	5	0·2
Medical patients:			
(*a*) Recent coronary occlusion	19	19	100
(*b*) Congestive cardiac failure	39	38	97
(*c*) Cerebral vascular accident	10	7	70
(*d*) Thrombotic states	10	10	100
(*e*) Pulmonary tuberculosis	38	4	10
(*f*) Thyreotoxicosis ..	5	0	0
(*g*) Other medical conditions	173	76	44
Surgical patients:			
(*a*) Acute sepsis ..	12	12	100
(*b*) Chronic sepsis ..	30	27	90
(*c*) Urological conditions, patients in bed ..	44	37	84
(*d*) Fractures	43	33	77
(*e*) After operation ..	131	53	40
(*f*) Thyreoidectomy ..	7	0	0
(*g*) "Clean" hernia ..	19	0	0

For the purpose of the survey the following groups were selected: (i) normal subjects, (ii) patients suffering from medical diseases, (iii) patients suffering from surgical diseases, (iv) pregnant women.

[1] Working with grants from the National Health and Medical Research Council of Australia.

Technique.

Blood (4·5 millilitres) was collected from each subject by venepuncture, and immediately transferred to a tube containing 0·5 millilitre of 1·1% sodium oxalate solution. If frothing occurred during collection, another specimen was obtained. This was centrifuged and the supernatant plasma was pipetted off.

To 0·5 millilitre of plasma two to four drops of the reagent (β naphthol in 50% strength alcohol, through which air had been passed until the solution turned brown) were added until opalescence first appeared. This is a slight modification of the fibrinogen B test as described by Lyons.[1] It will be further discussed by Lyons in a later publication.

After the mixture had stood for ten minutes the presence of an obvious gel was regarded as a positive result for the purpose of the survey.

Results.

The results are set out in Tables I and II.

From this survey the following figures may be of some significance (Table III).

TABLE III.

Condition.	Number of Subjects.	Number with Fibrinogen B.	Percentage Occurrence of Fibrinogen B.
Normal condition ..	2,240	9	0·4
Thyreotoxicosis	5	0	0
Pulmonary tuberculosis ..	38	4	10
Sepsis	42	39	93
Recent coronary occlusion	19	19	100
Congestive cardiac failure	39	38	97

Discussion.

In the majority of cases of sepsis, recent coronary occlusion and congestive cardiac failure, fibrinogen B was found to be present. In addition, in the greater number of these conditions it was present in maximal amounts.

In normal subjects and in patients suffering from thyreotoxicosis or from pulmonary tuberculosis, fibrinogen B was rarely found and, when detected, was only minimal in amount.

It is interesting to note that the results obtained in the cases of sepsis and thrombotic states are consistent with the previous work of Lyons.[1,2,3] The apparent significance of the results obtained in cases of coronary occlusion has stimulated further investigations, which will form the subject for a later publication by Dunn and Lyons.

At this stage no attempt is made to draw any conclusions from this preliminary survey.

Summary.

A survey to ascertain the presence or absence of fibrinogen B in human plasma is being conducted.

This initial report includes 2240 normal subjects, 200 pregnant subjects and 580 patients who were suffering from various diseases.

The figures are presented, but no conclusions are drawn.

Acknowledgements.

We are indebted to Professor H. R. Dew, of the Department of Surgery, University of Sydney, for his invaluable help, and to the members of the medical, surgical and obstetrical staffs of the Royal Prince Alfred Hospital, who have made available patients for investigation.

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ILLUSTRATIONS TO THE ARTICLE BY Dr. P. W. VERCO.

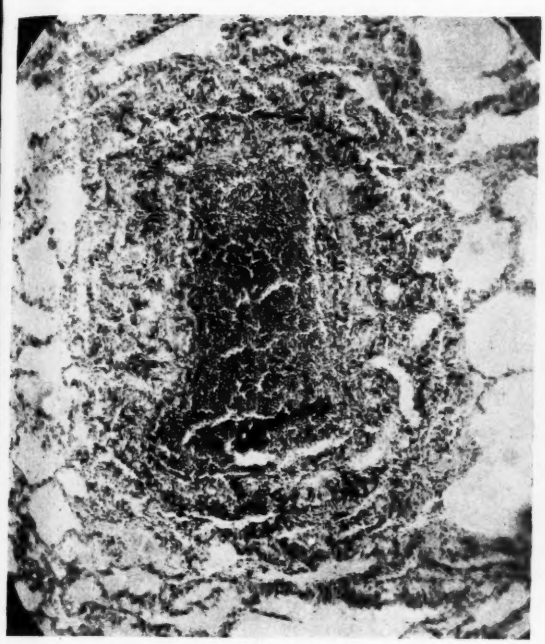

Figure I.

Frozen section (hæmatoxylin and eosin stain, × 40); Case 108. Bronchiole containing polymorphonuclear exudate and epithelial debris. Infiltration of bronchiolar wall and peribronchiolar tissue with lymphocytes, monocytes and plasma cells. Some alveolar walls show infiltration.

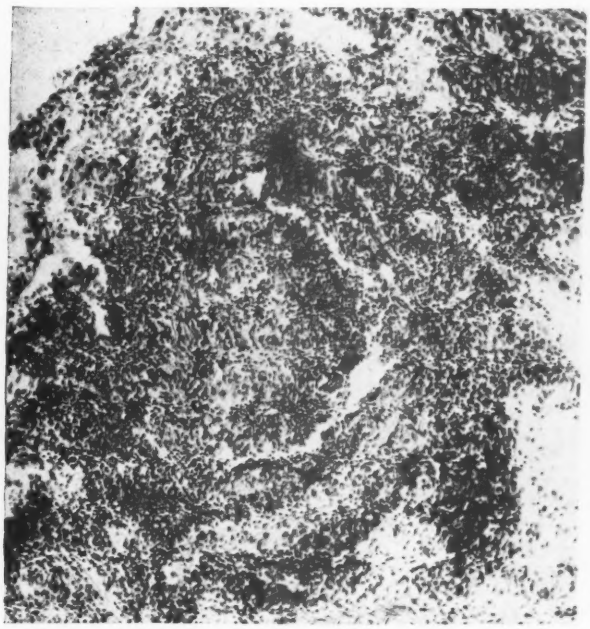

Figure II.

Frozen section (hæmatoxylin and eosin stain, × 80); Case 108. Commencing organization of bronchiolar plug, which contains a few polymorphonuclear cells and mainly lymphocytes, macrophages and fibroblasts. Œdema and infiltration of connective tissue surrounding bronchiole.

ILLUSTRATIONS TO THE ARTICLE BY Dr. A. R. HAMILTON.

Figure I.

Figure II.

ILLUSTRATIONS TO THE ARTICLE BY DR. MALCOLM FOWLER AND DR. E. L. FRENCH.

FIGURE II.

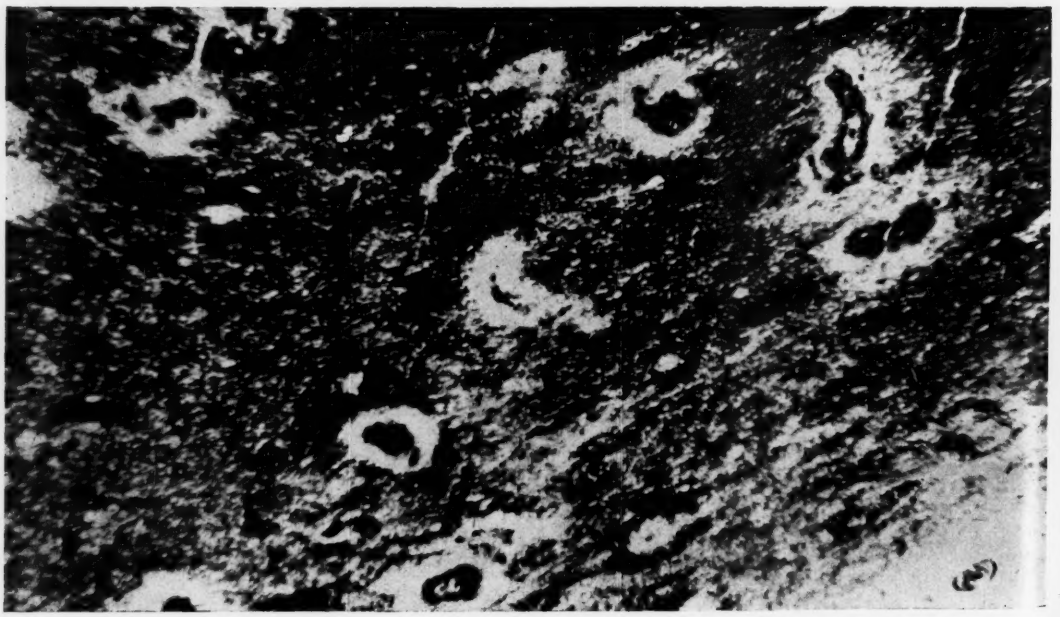

FIGURE III.

Reports of Cases.

A CASE OF ACUTE HÆMORRHAGIC LEUCOENCEPHALITIS (HURST) FOLLOWING ATYPICAL PNEUMONIA, WITH A DESCRIPTION OF CULTURAL AND VIRUS STUDIES.

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AND
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IN 1942 E. Weston Hurst[1] described an acute disease of the central nervous system characterized by severe damage to the walls of the blood vessels of the white matter of the brain. Perivascular necrosis, perivascular and focal demyelination, hæmorrhage and œdema were other conspicuous features. To this condition he gave the name "acute hæmorrhagic leucoencephalitis".

The present case is recorded not only because the disease involved the optic tracts and was associated with atypical pneumonia, but also because an opportunity was afforded us of conducting cultural and virus studies on the autopsy material. Previous descriptions have been confined to the clinical and pathological features of the disease.

CLINICAL RECORD.

A woman, aged thirty-seven years, was admitted to the Royal Adelaide Hospital on October 13, 1946, under the care of Dr. A. R. Southwood, with a five days' history of a "cold on the chest", causing difficulty in breathing and a cough with yellowish sputum. On the night before her admission to hospital she discovered that she could not use her left arm and that her left leg was weak. Sleep had been poor from coughing and shortness of breath.

On examination of the patient, she was breathing rapidly with an expiratory grunt. Her chest moved poorly, and on auscultation numerous rhonchi and moist râles were heard all over the lungs. The blood pressure registered 140 millimetres of mercury, systolic, and 85 millimetres of mercury, diastolic, the heart rate being 104 per minute.

A left-sided facial paralysis of the upper motor neuron type was present, and her tongue deviated to the left side. No ocular abnormality was detected. The left leg was weaker than its fellow, and was anæsthetic to light touch and pressure. Associated with exaggerated knee and ankle jerks on the left side was an extensor plantar reflex. There was a flaccid paralysis of the left arm, which was anæsthetic to light touch and pressure.

Nine hours after her admission to hospital the patient became semi-comatose, with stertorous respirations. The cerebro-spinal fluid was slightly cloudy and at a pressure of 120 millimetres of water. It contained five erythrocytes and 220 leucocytes (95% polymorphonuclear leucocytes and 5% lymphocytes) per cubic millimetre, with 125 milligrammes of protein and 100 milligrammes of sugar per 100 millilitres. On the following day (October 14, 1946) the cerebro-spinal fluid contained five red blood corpuscles and 1300 leucocytes (95% polymorphonuclear leucocytes and 5% lymphocytes) per cubic millimetre; the chloride content was 710 milligrammes per 100 millilitres. The leucocytes in the blood numbered 8500 per cubic millimetre. The report on the X-ray film of the chest taken on this day read as follows: "Lessened translucency at the left base, but no definite consolidation is detected".

The patient died in coma on the next day (October 15, 1946). Figure I shows the pulse and respiration rates and the temperature chart during the period of observation.

POST-MORTEM EXAMINATION.

The autopsy was performed by Professor J. B. Cleland twenty-three hours after the patient's death.

Macroscopic Examination.

The body was that of a well-nourished female.

Lungs.

The lungs did not collapse when the chest was opened, and both contained purple, sunken, airless patches studded with pale, firm spots resembling miliary tubercles. The intervening portion of the lung was pale and emphysematous. On the left side three-quarters of the lower lobe and one-third of the upper lobe were affected. On the right side only one-third of the lower lobe was involved. The congested bronchi contained mucopus. The bronchial and tracheal glands were large and œdematous.

Brain.

Damage to the brain mainly involved the white matter. Of the cerebral hemispheres, the right contained the more extensive lesions, being swollen and larger than the left. The middle third of the *centrum semiovale* of the right

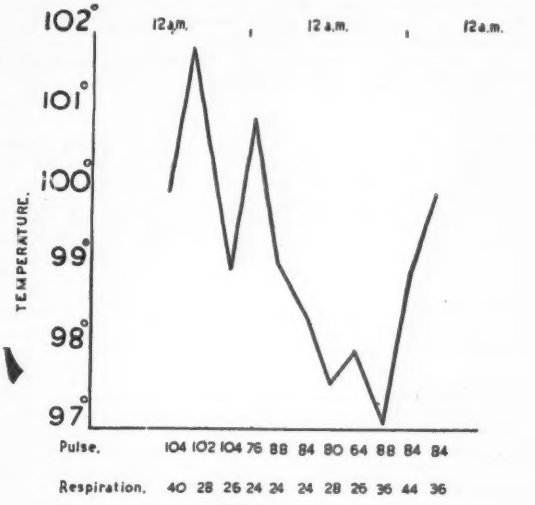

FIGURE I.
Pulse and respiration rates, and the temperature chart.

side was soft and peppered with hæmorrhagic spots; after fixation, examination of these punctate spots with a hand lens revealed that they were annular in shape with a whitish centre. The *centrum semiovale* underlying the parietal area of the cortex contained the most severe lesions on the left side, but they were more restricted in extent than those on the right side. The *corpus callosum* was affected. The disease did not involve the occipital or frontal white matter of either hemisphere. The lesions could be traced through the internal capsule and into the *basis pedunculi*, the most obvious changes again being on the right side. A few lesions were noted in the floor of the aqueduct in the mid-line. Examination of the superior and middle cerebellar peduncles revealed similar changes, extending into the white matter of the cerebellum and surrounding the dentate nucleus, and into the lateral walls, floor and roof of the fourth ventricle. The right side was swollen. The closed part of the medulla was not affected. The optic tracts of both sides were involved with equal severity. The spinal cord was not available for examination.

Other Organs.

Except for some congestion, the other organs were normal.

Microscopic Examination.

Lungs.

Sections of the lungs were taken through the dark nodular areas, and examination revealed extreme con-

gestion of the larger arteries and veins, of the submucosal and the extramuscular bronchiolar vessels and of the alveolar capillaries. No hæmorrhages were seen.

The walls of the bronchioles in these areas were infiltrated with plasma cells and a few lymphocytes, and their lumina were filled with polymorphonuclear leucocytes (Figure II). The epithelium had often sloughed, particularly when the exudate was necrotic. In many bronchioles this necrotic material was undergoing early organization, shown by the ingrowth of macrophages and fibroblasts.

Some of the alveoli were affected by the same process, which could be seen extending into them by direct continuity from the terminal bronchioles. A few giant-cells, of the Langhans type, were noted in areas of alveolar organization. Most of the remaining alveoli contained many macrophages, with a little serous fluid here and there, but only occasionally were there any interstitial changes in their walls.

The close association of areas of commencing organization with fresh foci of polymorphonuclear leucocytes, the two often immediately adjacent and well-demarcated from each other, was an unusual feature of both the bronchiolar and alveolar exudates (Figure II).

The subpleural vessels were intensely congested, but the pleural lining was unchanged.

Brain.

Transverse sections of the brain were taken through (i) the right cerebral hemisphere at the level of the geniculate bodies, (ii) the left cerebral hemisphere at the level of the *tuber cinereum* and (iii) the brain stem across the facial colliculi; the last-mentioned section included some of the cerebellum. These sections were embedded in celloidin and stained with hæmatoxylin and eosin, with hæmatoxylin and Van Gieson's stain, with Nissl's stain, and with Weil's myelin sheath stain. Frozen sections were stained for axis cylinders by Bielschowsky's silver method, and with *Scharlach R* for fat. In addition some paraffin blocks were made of various areas.

As in previous reports of this condition, the lesions found were of an acute nature, situated particularly around blood vessels of the white matter. Where the brain was most severely damaged most of the capillaries and tiny veins were surrounded by areas of necrotic tissue, sometimes quite extensive, or by small intracerebral hæmorrhages, mainly of the "ball" type. Sometimes there appeared to be a mixture of these two types of lesions. Polymorphonuclear leucocytes formed dense, wide collars in the perivascular spaces, from which they had spread into the surrounding brain substance. Necrosis of these cells was noticeable around the blood vessels and in their walls. Often the areas of necrosis and hæmorrhage were unassociated with any cellular reaction. Microglial cells, in different stages of development, appeared in varying numbers, and, here and there, made a densely-packed pattern, resembling a pavement. In these parts of greatest damage œdema was extreme, while in the Virchow-Robin spaces there was a thin exudate usually mixed with cellular elements. In some places it was seen unobscured, having a band of polymorphonuclear leucocytes between it and the vessel wall. Occasionally the fluid and the cells were confined to one side of a vessel with normal brain on the other side. In areas of perivascular necrosis the neuroglia had disappeared or were pyknotic. Here, too, there was some beading of the nerve fibres.

Perivascular demyelination, often partial, occurred with perivascular necrosis, but it was also present alone, in equal intensity, with only a reaction of polymorphonuclear leucocytes and scavenger cells. Where microscopical lesions were most severe, there appeared, on macroscopic examination of the stained tissue, to be diffuse partial destruction of myelin—an appearance produced mainly by œdema and cellular elements separating the nerve fibres, and so reducing the density of the tissue. Some concurrent demyelination and much ballooning of the sheaths were, however, present in these œdematous areas.

Faintly-staining fat droplets were present in the scavenger cells, particularly in those near blood vessels. Many pale violet bodies resembling *corpora amylacea* were present in the white matter of severely affected areas. Examination of the capillaries and venules revealed swollen endothelium, while in a few places their walls were necrotic. No thrombi were seen. The meninges contained scattered polymorphonuclear leucocytes.

The microscopical damage ran *pari passu* with that seen with the naked eye, but extended beyond it for a variable degree in the form of perivascular demyelination with cellular elements, and further out still as infiltration with polymorphonuclear leucocytes. Compared with the *centrum semiovale*, the cerebellar peduncles, especially the right, contained a greater proportion of perivascular necroses, while hæmorrhages were less obvious. A few of the fibres of the lateral part of the pons were destroyed.

The "U" fibres of the *centrum semiovale* were spared both demyelination and the more acute changes; but polymorphonuclear leucocyte infiltration was often pronounced. In the optic tracts the damage was of a predominantly acute nature with hæmorrhages (often ring-shaped), necrosis, and unusually wide zones of perivascular demyelination (Figure III).

The grey matter was not involved, except by extension from foci in the adjacent white matter, usually in œdematous areas. For example, the cells of the dentate nucleus were severely affected, and showed chromatolysis, nuclear eccentricity, convexity of cell outline and dissolution into "ghost" forms.

Many different pathological pictures resulted from a combination of the various degrees of cerebral damage and cellular reaction. For a detailed description of these effects Hurst's original article may be read.

BACTERIOLOGICAL AND VIRUS INVESTIGATIONS.

A portion of the lungs showing purple, airless patches, and pieces of the brain from the right and left occipito-parietal junctions, were obtained with aseptic precautions. Attempts at aerobic and anaerobic cultures from these specimens produced no growth after forty-eight hours' incubation at 37° C.

A suspension of lung was prepared by grinding the tissue with sterile sand. One millilitre of this suspension was injected subcutaneously into each of two guinea-pigs. Eight weeks later these animals were killed, and an autopsy failed to reveal any evidence of tuberculosis or other abnormality.

Further animal inoculations were performed in an effort to isolate a virus. The suspension of lung was centrifuged at approximately 2500 revolutions per minute for fifteen minutes to remove coarse particles, and a portion of the supernatant fluid filtered through a gradocol membrane of average pore diameter of 600 millimicrons. The filtrate was inoculated into two rabbits, two guinea-pigs and six mice intracerebrally, and into six mice intranasally. The rectal temperatures of the rabbits and guinea-pigs were taken daily, and over a period of twenty-one days the two rabbits had normal temperatures. One guinea-pig was moribund on the sixth day after injection with the lung filtrate. Its temperature was below 96° F., and the head was retracted and the neck rigid. The animal was killed with chloroform and the brain removed aseptically. A transverse section through the mid-brain was fixed in Zenker fixative and paraffin sections were prepared. Examination of sections stained with hæmatoxylin and eosin revealed no abnormality. The abdominal and thoracic viscera appeared to be normal, and an attempt at culture of microorganisms from the heart blood was unsuccessful. A portion of the brain was ground with sterile sand and an emulsion in saline solution was made. Attempts at aerobic and anaerobic cultures of the emulsion were unsuccessful, and the emulsion was inoculated into four more guinea-pigs, two intracerebrally and two intranasally, with negative results, the animals being observed for one month.

The second guinea-pig of the pair receiving the original lung filtrate died on the eleventh day after inoculation. Post-mortem examination failed to disclose any abnormality. Passage of brain substance into further guinea-pigs met with negative results. The mice inoculated intracerebrally and intranasally all survived for one month without showing any symptoms. Two rats that

TABLE I.

Case Number.	Authors.	Sex of Patient.	Age. (Years.)	History.	Duration of Illness from Onset of Cerebral Manifestations.	Post-Mortem Findings Apart from Central Nervous System.	Cultural Studies.	Case Précis.
I	Hurst.	F.	33	Bronchopneumonia three months before.	42 hours.	—	Not undertaken.	Acute onset of giddiness and unilateral weakness with headache. Speech failed. Could swallow, but not chew. Mental confusion on admission to hospital; right flaccid paralysis of face, tongue, arm and leg. Temperature 100° to 101° F.
II	Hurst.	M.	33	"Woodside throat" lasting ten days, three weeks before.	6 days.	—	Not undertaken.	Collapsed at work, and vomited; afterwards capable only of slow speech. Walked two and a half miles. Pyrexia noted at hospital. Next two days in bed; headache, "hot and shivering". No neurological signs present. Felt better next day, but still mentally retarded on the following day, when he had an epileptic seizure with coma in the evening. No localizing nervous signs. Temperature 97·6° to 99·6° F.
III	Henson and Russell.	M.	21	Febrile illness lasting five days, ten days before.	4 days.	Inhalation bronchopneumonia. Nasopharyngitis with accessory sinusitis. Severe parenchymatous degeneration of kidneys.	Not undertaken.	Headache and numbness of right arm. Next day a moderate right hemiparesis. Drowsy and slow in answering questions. Vomited. Next day weakness of left arm noticed with spastic right hemiplegia. Incontinence of urine. Next day spastic quadriplegia with decerebrate movements, followed by flaccid quadriplegia. Urine solid with albumin, and casts present. Temperature 102·8° to 104·2° F.
IV	Shallard and Latham.	F.	7	Cold and cough for three days before admission to hospital.	5 days.	—	Not undertaken.	Vomited after evening meal; listless and feverish next day, and became limp. Next day lost use of limbs, especially right arm and left leg. On admission to hospital constant movement of the eyes. Right arm paretic and right leg flaccid. Left side unaffected. Pupils unequal. Temperature 97·8° to 99·6° F.
V	Wolf and Cowen (information in writing).	F.	6	Whooping cough vaccination started five weeks before death.	48 hours.	?	?	After the fourth injection of whooping cough vaccine she developed asthmatic attacks. Convulsions 48 hours before death.
VI	Fowler and French.	F.	37	Five days' cough and dyspnœa.	2·5 days.	Atypical pneumonia.	Attempt unsuccessful.	Acute onset with paralysis of left arm and weakness of left leg. On examination, rapid respiration with moist sounds in the chest. Left-sided facial and lingual paralysis. Flaccid paralysis of left arm and weakness of left leg with left hemianæsthesia to light touch and pressure. Temperature 97·1° to 101·8° F.

had received lung filtrate intracerebrally also remained normal for one month.

While obtaining specimens of the brain from the subject at the post-mortem examination, one of us (E.L.F.) noted that the red blood corpuscles exuding from the cut surface of the brain clumped in the dish on standing at room temperature. Examination of these corpuscles under the microscope showed them to be agglutinated and not in rouleaux. The brain was emulsified as before and a similar range of animals was inoculated intracerebrally and intranasally as for the lung filtrate. Temperatures were taken daily over a period of three weeks in the rabbits and guinea-pigs, and in all cases were normal. None of the animals inoculated with the brain emulsion developed any detectable abnormal symptoms during two months' observations.

In addition to animal inoculation tests, developing check embryos were inoculated intraamniotically and on to the chorio-allantoic membrane by the technique of Beveridge and Burnet.[2] The brain emulsion was lightly centrifuged and inoculated directly, while the lung filtrate was used as another inoculum. Unfiltered lung emulsion was mixed with penicillin and sulphadiazine and inoculated into eggs, as recommended for the direct isolation of influenza virus from throat washings by Burnet.[3]

All the eggs were carried through six passages without the discovery of any evidence of infection.

DISCUSSION.

A summary of the relevant findings in the five previously reported cases[1][5][6][7] and in the present case is presented in Table I.

In our case the three main points of interest, all hitherto unreported, are (i) the occurrence of atypical pneumonia, (ii) the presence of lesions in the optic tract, and (iii) the bacteriological and virus studies of the brain.

The clinical picture produced by the lung lesions was that of bronchiolitis, and this was borne out by the histological examination. Golden's[4] study of 42 cases enabled him to describe certain common pathological features to those cases diagnosed clinically as atypical pneumonia. In summary, they are acute bronchiolitis with infiltration with polymorphonuclear leucocytes, and desquamation of the bronchiolar epithelium into the lumen, which is usually dilated. Further features include infiltration of bronchiolar walls with plasma cells and lymphocytes, which extends into the interstitial pulmonary tissue, the alveoli occasionally containing a few polymorphonuclear leucocytes. One of the most important findings in his cases was the absence of organisms in sections suitably stained or on attempted culture. Our case resembled this picture closely, although interstitial changes in the alveolar walls were minimal.

The appearances in our case suggested that there had been two phases of inflammation in the lungs. The necrotic exudate of the first phase was undergoing organization when a fresh wave of infiltration with polymorphonuclear leucocytes, together with much hyperæmia and some fluid exudate, manifested itself. Although in the other reported cases of acute hæmorrhagic leucoencephalitis histories have been given of antecedent febrile illness occurring from

three months to a few days before the onset of neurological disease, no definite pathological lesions were present at the post-mortem examination which could be correlated with the encephalitis except in Henson and Russell's[6] case, in which naso-pharyngitis with sinusitis and tubular degeneration of the kidneys were found. In Wolf and Cowen's case a history was given of consistent hypersensitivity to various substances since birth. The following are Dr. Wolf's own words (from a private communication in writing):

> Whooping-cough vaccine was administered five weeks before death, a first course having been given a few weeks previously. After the fourth injection, she developed severe asthmatic attacks associated with convulsions 48 hours before death.

In 1923 Weinberg[8] reported, in cases of severe anaphylactic shock, perivascular infiltration, hæmorrhage, occasional thrombosis, diffuse degenerative changes in nerve cells and alterations in the myelin sheaths. By giving repeated injections of aqueous emulsions of alcohol-ether extracts of sterile normal rabbit brain, Rivers and Schwentker[9] produced pathological changes accompanied by hæmorrhage and myelin destruction. Ferraro[10] has likened these lesions produced by Rivers and Schwentker to an Arthus phenomenon localized in the white matter, and has suggested that the primary demyelinating diseases may have their basis in the combination of antigen and antibody in the brain. More recently a number of investigators[11][12][13] working independently found that normal monkey or rabbit brain, injected subcutaneously with adjuvants using Freund's[14] technique, produced acute disseminated encephalomyelitis. These experimental lesions bear a striking resemblance to those seen in Hurst's two original cases and in the present case.

The recent work of Burnet's school[15][16] dealing with the enzyme action of influenza virus on cell surfaces is possibly not unrelated to this problem. After contact with one of the influenza-mumps group of viruses the antigenic pattern of cells is changed. Moreover, the action of the virus enzyme is to release "split products" from the receptor sites of the cell. Such cells or "split products" will be treated as foreign material, and, if antigenic, will evoke the production of antibodies. A somewhat similar mechanism has been suggested by Gear[17] to explain the intravascular hæmolysis occurring in blackwater fever, the infected erythrocytes becoming auto-antigens and producing antibodies which sensitize the infected cells to the action of complement. Although in our case an infective agent was not isolated (possibly because all the animals used belonged to a non-susceptible group), it is reasonable to assume that the pneumonia was caused by a micro-organism, most probably a virus. Could not this infection of the lungs have provided the antigenic stimulus in the form of antigenically altered bronchiolar cells, or "split products", due to virus enzyme action? The patient had the lung infection for at least five days before the cerebral symptoms appeared. During this period antibodies formed could become "fixed" to various cells, and then further antigenic material reaching the "fixed" antibody may have produced an allergic reaction. The different chronological processes in the bronchioles suggest that the brain was not the only organ to suffer a reaction, and that the lungs may react similarly at the original site of entry of the antigenic stimulus. This mechanism is essentially similar to that suggested by a number of workers to explain the experimental lesions following subcutaneous injections of brain tissue with adjuvants.

The involvement of the optic tracts in other demyelinating diseases, particularly in disseminated sclerosis and in Devic's disease, is well known. (See Hurst, de Crespigny and Fry,[18] Balser,[19] and Silbermann.[20]) Hurst[21] found patches of demyelination in the optic tracts of monkeys (beside lesions elsewhere in the brain) after repeated injections of sodium azide and potassium cyanide. Kabat[11] and his co-workers produced a picture like disseminated encephalomyelitis in man, in some cases with extensive areas of demyelination in the optic tracts, after the injection of monkey or rabbit brain tissue into monkeys. Hurst's suggestion that acute hæmorrhagic leucoencephalitis provides a link between the hæmorrhagic encephalitides and the demyelinating conditions finds support in the involvement of the optic tracts in this case.

SUMMARY.

A case of acute hæmorrhagic leucoencephalitis associated with atypical pneumonia is described in a woman, aged thirty-seven years. She presented with a five days' history of "cold on the chest", developed left hemiparesis and hemianæsthesia, and died in coma two days after her admission to hospital. The clinical and pathological features of the case are compared and contrasted with five previously reported cases in the literature. The involvement of the optic tracts in the present case is a new feature of this condition.

Bacteriological studies and animal and chick embryo inoculations for virus failed to detect an infective agent in the brain or lungs of the patient.

The possible relationship of the atypical pneumonia to the lesions in the brain is discussed in the light of recent work dealing with the modifying influence of virus on the antigenic structure of cells.

ACKNOWLEDGEMENTS.

Our thanks are due to Dr. A. R. Southwood for permission to publish the clinical notes of this case. We are indebted to Professor J. B. Cleland for the pathological material and the opportunity to obtain suitable material for cultural examination and virus studies, and to Dr. Charles Swan for encouragement and advice in the preparation of this report. We wish to thank Mr. Noel Peake, of the Department of Anatomy of the University of Adelaide, for the photomicrographs.

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Legends to Illustrations.

FIGURE II.—Showing a bronchiole containing inflammatory exudate which is organizing above, with a fresher exudate of polymorphonuclear leucocytes below. (Hæmatoxylin and eosin; x 250.)

FIGURE III.—Extensive perivascular demyelination in the optic tract. (Weil stain; x 30.)

AN UNUSUAL DISLOCATION.

By A. R. HAMILTON,
Sydney.

IN view of the extreme rarity of the occurrence, I consider that a brief history of the following case is worth recording.

Clinical Record.

W.R., a male patient, aged fifty-one years, was a dogman by occupation. On August 26, 1946, he was admitted to hospital with numerous lacerations and a fracture of his left femur and a lateral and forward dislocation of his left *os calcis* without any associated fracture of the foot or ankle (Figure I). The injuries had been caused by a fall of two tons of timber onto the anterior aspect of the leg whilst he was in a half-upright position, the leg being thus forced backwards against his fixed heel. The reduction of the *os calcis* was readily effected by manipulation without recourse to skeletal traction (Figure II) and the foot was encased in a plaster boot for nine weeks. The fractured femur was treated in a Thomas splint with skin traction. The abrasions on the foot became rather septic, and this resulted in considerable stiffness of the toes.

On examination of the patient on June 20, 1947, his ankle, subtaloid and mid-tarsal movements were painless and practically full, although the toes were still rather rigid. Some disuse mottling was still present.

Acknowledgement.

I wish to acknowledge the assistance given by the X-ray staff of Balmain Hospital in the production of the films in this case.

A CASE OF GOLD DERMATITIS SUCCESSFULLY TREATED WITH BAL.

By EGON SUERTH,
Beaconsfield, Tasmania.

MRS. L.B., aged fifty-three years, had a history of rheumatoid arthritis of moderate severity for the past six years. X-ray films showed bony changes suggestive of atrophic arthritis involving her left sacro-iliac joint and both hands. Her blood sedimentation rate (Westergren) was 20 millimetres in one hour at the beginning of the treatment in November, 1947. Her hæmoglobin value was 90% and her white blood cells numbered 8000 per cubic millimetre.

She was considered a suitable subject for gold therapy, and treatment commenced on December 10, 1947. "Solganol B Oleosum" was given by intramuscular injection biweekly in doses of 10, 25 and 50 milligrammes, four injections of each dose. Thereafter a weekly injection of 100 milligrammes was given until a total of 0·64 gramme was reached on February 11, 1948.

During this course no "protective" preparations, such as vitamin *C* or liver extract, were given. Prior to each injection the urine was tested, the skin inspected, and the patient questioned with regard to any itch. Her white blood cells were counted at fortnightly intervals, the last count five days prior to her final injection on February 11, 1948, revealing a total of 6000 white cells per cubic millimetre, the differential white cell count and the number of red cells were normal.

On February 15, 1948, without any warning, generalized severe pruritus set in. Maculo-papular eruptions appeared on her trunk and extremities, leaving the face, neck and mucous membranes free. No lesions were detected on the gums or throat. The patient was distressed, prostrated and extremely restless. The temperature and pulse rate were normal. The urine output was satisfactory, and no albumin was present.

On February 16, 1948, the patient was admitted to hospital. On her admission her hæmoglobin value was 82%, her red cells numbered 3,900,000 and her white cells less than 2000 per cubic millimetre. Unfortunately no differential white cell count or enumeration of platelets was carried out at this stage. The pruritus was more pronounced than on the previous day. The rash, although deeper in colour and more distinct, had not spread further. The skin was hot to touch, not not œdematous. There was no weeping or exfoliation, or any involvement of the buccal mucosa or tonsillar region.

The diagnosis of toxic reaction to gold (dermatitis and leucopenia) was made.

Treatment consisted in sedation, the forced administration of fluids, and the giving of calcium gluconate by intravenous injection and "Campolon" in massive doses. In addition, in view of the possibility of agranulocytosis, penicillin was given by four-hourly intramuscular injection. Calamine lotion was applied topically. This treatment was continued for five days with no visible change of her general or local condition.

On February 21, 1948, an urgent request for BAL was sent to the Royal Melbourne Hospital. The drug was received here on the same day.

BAL injections were commenced on February 22, 1948, in four-hourly doses. The temperature was checked before each injection. Any other treatment was discontinued and only copious fluids were given by mouth. A rise of temperature occurred on the second day (99·2° F.) and no BAL was given on that day. On the third day full treatment was resumed and it was completed without ill effect or further rise of temperature on February 27, 1948.

The response to BAL was in brief as follows. On February 24, 1948, the pruritus and skin condition were unchanged. Examination of the blood showed a rise in the number of white cells from less than 2000 per cubic millimetre on February 16 to 72,000, the proportion of eosinophile cells being 20%; other white cells were of normal types and frequency. On February 25 there was much diminution of the pruritus associated with some blanching of the rash; the general condition was improved. On February 27 there was no pruritus; the skin looked healthy, with slight scaling; the patient felt well and wished to get up. On March 8 she was discharged from hospital after an uneventful recovery.

Six months later this patient showed a great improvement of her rheumatoid arthritis and her blood sedimentation rate had returned to normal.

Comment.

1. No reliable subjective or objective signs exist at the present day to anticipate toxic reaction to gold. Precautions taken in the case described above proved fruitless. It is doubtful whether the fact that this patient gave evidence of allergic manifestations in the past (she was a

sufferer from hay fever) was justification enough to withhold gold treatment.

2. BAL brought about a dramatic response in this case of gold intoxication, which was characterized by dermatitis and leucopenia, after previous routine methods had failed.

3. More evidence has to come forward in regard to the effect of BAL in cases of gold dermatitis before this new drug can be evaluated properly in this condition. A trial of BAL in granulocytopenia and thrombocytopenia due to gold might be worth while.

4. If its action should prove specific its use would counteract the fear which is always present in anyone who applies chrysotherapy in rheumatoid arthritis—a fear which to a great extent is responsible for the fact that gold has been abandoned by many American clinicians and for the trend in Empire countries to use this powerful drug in fractional doses only.

Acknowledgements.

I wish to thank Dr. J. L. Grove, Launceston, for his encouragement, and the medical superintendent of the Royal Melbourne Hospital for providing the BAL.

Reviews.

MEDICINE, SCIENCE AND POSTAGE STAMPS.

MEDICAL MEN, probably above all others, are prone to have a hobby, and those who are interested in the royal hobby of philately will find much to interest them in a small volume entitled "Medicine and Science in Postage Stamps", by W. J. Bishop and N. M. Matheson.[1]

All sorts of collections of postage stamps illustrating such topics as birds, animals, architecture or even ships, have been made and reported upon from time to time. Any of these may lead the collector into a variety of interesting fields of investigation, but this is the first we have seen in which medical men and men of science as depicted on postage stamps have been reviewed.

The collectors point out some rather surprising gaps, suggesting that they are probably due to the rather rigid postal traditions of Great Britain and America. It may be, of course, that the chauvinistic attitude of some continental countries explains the predominence of this type of commemoration stamp on the continent of Europe, and the fact that some rather unimportant people have thus been honoured. We can only hope that before long such giants as Harvey, Lister, Jenner, Virchow, Helmholtz and Darwin will be honoured in this way and again brought before a world which so easily forgets.

The authors describe their own very fine collection, give excellent illustrations, a good bibliography (which is remarkably extensive for a subject like this) and a full descriptive catalogue at the end. This small book is therefore one which will make a welcome addition to the library of any stamp collector.

INTESTINAL OBSTRUCTION.

"ACUTE INTESTINAL OBSTRUCTION", by Rodney Smith, provides interesting and instructive reading.[2] The sections on clinical and radiological diagnosis and on intestinal decompression under radiological control are particularly good. The injection of a "solution" (*sic*) of barium sulphate when the Miller-Abbott tube has reached the site of obstruction and the study of the mucosa by the spot film method are an interesting suggestion.

For those cases in which the Miller-Abbott tube fails to pass the pylorus the author suggests laparotomy and gastrostomy. But, if the tube was inserted through the mouth or nose, the laparotomy without the gastrostomy would probably suffice.

Surely the old conventional danger angle at the mesenteric attachment can be eliminated by separation of the mesentery from the bowel before the end-to-end anastomosis is made.

In the section on fluid balance there is no description of the principles which govern the ratio between water and sodium chloride.

The credit for developing aseptic anastomosis is given to Martzloff instead of to Seton Pringle and Jan Schoemaker.

Australian surgeons are repeatedly disappointed in reading books, published overseas (such as this one), by the scanty recognition of the principles of treating intussusception which were established by the late Sir Charles Clubbe, of Sydney. In spite of their unequalled results the methods have not been generally accepted. This is in curious contrast to the ready acceptance of each successive method of intubating the other end of the alimentary tract. It seems that the readiness with which an advance in medicine is recognized and accepted depends, not only upon its intrinsic merit, but also upon its place of origin. In this respect, workers in far-flung outposts are at a disadvantage.

Nevertheles, the book is stimulating and well worth reading.

RADIOTHERAPY.

RADIOTHERAPISTS have been aware for some time that a text-book of radiotherapeutic practice was in course of preparation in Manchester. This book, "The Treatment of Malignant Disease by Radium and X-rays", by Ralston Paterson, has now come to hand.[1] It is written for the practising radiotherapist and the post-graduate student. It omits clinical and pathological considerations of the diseases described, and deals solely with the practical details of radiation treatment.

Initial chapters describe the principles of radiation treatment and the choice of treatment apparatus and factors. The Manchester school pioneered the use of small field beam directed X-ray therapy, and J. L. Dobie has contributed an outstanding chapter on the principles involved and the methods adopted to ensure their application. The methods adopted of arrangement of radium sources for interstitial or superficial application as elaborated by Paterson and Parker are redescribed. These have already been adopted throughout British centres and are gradually coming into use throughout the medical world. The elaboration of this system undoubtedly constituted the greatest single advance in the practice of radium therapy and enabled radium dosage to be prescribed, as is X-ray dosage, in terms of *r* units.

The subsequent chapters deal with the diseases amenable to irradiation in the different anatomical sites. These chapters detail the indications for the various forms of treatment, the methods to be adopted and the results to be expected. This advice is given as the result of the experience of the author and his collaborators at the Manchester Institute, and represents the standard practice of the school. Not every radiotherapist will agree with these recommendations in their entirety, but it would be idle to offer minor criticisms. In few other centres has radiotherapy had such excellent opportunities of proving its value, and the advice conveyed in these pages represents the considered opinions of thoroughly experienced clinicians.

Chapters on organization, records and the protection of staff personnel from radiation hazards complete the author's work, and the concluding chapter is contributed by Dr. Edith Paterson. A fine review of recent work on the biological effects of X and γ radiations is contributed, and this chapter gives a most valuable summary of the problems involved, the line of research attack which has been adopted, and the deductions which at present appear justified.

This text-book constitutes a very essential addition to the possessions of every practitioner of radiation therapy. It has been eagerly awaited and it is assured of a great welcome. The author and his colleagues are deserving of praise and thanks for the work which has gone into the volume, much of which has been done during the latter war and the even more difficult post-war years. The book is beautifully printed and bound, and the many illustrations are excellently produced.

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[1] "Medicine and Science in Postage Stamps", by W. J. Bishop, F.L.A., and N. M. Matheson, F.R.C.S.; 1948. London: Harvey and Blythe, Limited. Distributors: H. K. Lewis and Company, Limited. 7¼" × 4", pp. 86, with illustrations. Price: 7s. 6d.

[2] "Acute Intestinal Obstruction", by Rodney Smith, M.S., F.R.C.S., with a chapter of Radiological Diagnosis by Eric Samuel, M.D., F.R.C.S., F.F.R., D.M.R.E., and a foreword by Rupert Vaughan Hudson, F.R.C.S.; 1948. London: Edward Arnold and Company. 8½" × 5", pp. 274, with illustrations. Price: 18s.

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The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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UNIVERSITIES AND THEIR PROBLEM.

THE problems associated with the post-war overcrowding of universities which have been discussed on several occasions in these columns during the last few years have not been confined to Australia. They have arisen in every democratic country, and in Great Britain discussions and investigations have taken place. Generally speaking the origin of the problems may be described as twofold. In the first place during the war years many young men and women, who in the ordinary course of events would have become university undergraduates, were serving with the fighting forces or as members of the ancillary services. After the cessation of hostilities large numbers of these young people—some of them were then not so very young—wanted to make up for what they had lost in time and in pursuit of the knowledge that would fit them for their life's work. A second origin, itself no doubt partly the result of the war, lies in the progress that had taken place in social evolution, as a result of which university education is being made available to an ever-increasing number of young persons. It may be that in the last respect Australia is somewhat in advance of the Old Country, but if that is so, it does not make the task of planning for the future any less difficult. Great Britain has the habit of appointing commissions or committees to inquire into matters of this kind; sometimes bodies such as the British Medical Association and the Royal College of Physicians of London appoint committees to the task. While possibly a great deal of such effort does not produce immediate results that may be credited to a particular effort, there is no doubt that the opinion of people concerned, and indeed public opinion, is influenced in this way. Unfortunately in Australia there is still too great a tendency to rely on inquiries made in Great Britain and other countries, and to pass by an opportunity to find a way or to throw light on a difficulty with a shrug of the shoulders and a muttered "what is the good?". This does not imply that Australia should rely solely on her own efforts in the solution of certain problems; it means that she should make her contribution instead of being content to allow others to make all the effort. But these are observations made as it were in passing. The present object is to draw attention to an investigation recently made by Nuffield College into "The Problem Facing British Universities", and published in pamphlet form.[1]

The pamphlet is the work of the Education Subcommittee which was set up originally in connexion with the Social Reconstruction Survey undertaken by Nuffield College; it is as a matter of fact the fifth statement that the subcommittee has made. It is not a report in the sense that the authors comply with terms of reference. The subject is discussed and the difficulties are dissected, but actual remedies are not recommended. Rather are possibilities suggested, some well-known ground is covered, and conditions are stated which must be fulfilled. Sometimes the course of action is clear; sometimes what is clear is the thing that should not be done. In this way the pamphlet, or the "statement" as it is called, is stimulating. To start with, it is shown that the demand on universities today comes from many quarters and is greater than it was before the last war. For example, the professions, which have always looked to universities, are asking for further provision for their needs; new professions are pressing for similar provision; industry, recently become conscious of its reliance on science, looks to universities for more trained scientists; governments and large corporations are calling for more trained economists and statisticians; expanded social services call for workers, of whom many need a university training; and lastly the country (Great Britain) has adopted a policy of extending adult education. In addition to all these demands there has been a large increase in the secondary school population. And in consequence there will be a further increase in the number of students who will expect to go to a university. At this stage it will be well to point out that the demand for university education is not the same in all countries; for this there are many possible reasons and these include the system and grading of schools, the social organization and the general attitude of people and perhaps what we may call the community habit. The proportion of the population which enters universities in Scotland is twice as large as in England. Again, compared with Great Britain, the United States with three times the population had in 1940 thirty times the number of full-time college and university students. Perhaps these facts are a reflection of the availability of university education or a community habit or both; he would be a bold man who would declare that university attendance was an indication of the educational standards or intellectual attainments of a country. Anyone inquiring into this subject will naturally ask what is the purpose of a university. The subcommittee states that the business of a university is the education of its members and adds that if professional training is consistent with a full education, by all means let them be combined, but education comes first. Later on it declares that just because universities have to assist in the education of certain professions, "they have constantly to safeguard the general character of the education they give, the balanced and comprehensive approach to learning and research for which they stand, against excessive specialization". However specialized the outcome is to be, "there is always an element of general education or culture in the teaching a university gives; . . . this teaching is

[1] "The Problem Facing British Universities", Nuffield College; 1948. London: Oxford University Press. 8¼" x 5¼", pp. 132. Price: 5s.

given by scholars and scientists who are themselves working at the frontiers of knowledge". And here we may remark that if a university teacher is so overburdened with the demands on his time that he is unable to work "at the frontiers of knowledge", in other words to engage in research, his teaching will lack life, the student will suffer and the university will fail in its purpose. The subcommittee finds it necessary to stress the older claims upon universities, because they are in danger of being overlooked in the excitement associated with the discovery of new possibilities and the formulation of new demands. New demands have to be examined in relation to the work of universities as a whole. The question is asked: If the university takes a part in law, why not in accounting? If in forestry, why not in horticulture? If in engineering, why not in navigation? If in agriculture, why not in commerce? If in architecture, why not in building? If in music, why not in dramatic art? The distinction must always be drawn, we read, as it always has been in good university practice, by a consideration of the content of the specialized instruction sought. "If a profession wishes the study of the subjects peculiar to it to be associated with a study of the underlying sciences or other branches of knowledge . . . there is a case for including it in the university's scope." There are a number of professions to which certain scientific studies carried on in universities are not only relevant but fundamental, though their practice calls for actual experience which universities cannot give. The advancement of knowledge is the concern of the university, its application to human needs that of the learned professions. The line between the purely professional and the purely scientific is not clear; in practice it is blurred and similar work is done on both sides. The primary task of the university is the preparation of the mind of the individual. It must be frankly admitted that the great majority of occupations involve so restricted a range of theoretical or scientific knowledge that they can be taught just as effectively and much more economically in the occupation than in a university.

As practitioners of medicine, readers of this journal are interested chiefly in the medical school side of universities. All that has been taken from the Nuffield College Subcommittee's statement applies to medical schools in this country. It applies to medicine particularly because the scientific side and the practical or professional side are obvious, and over-emphasis on one at the expense of the other must prove fatal to the production of what are known as good doctors. There is, or should be, no need to insist on the broad bases of university education for doctors. Paraphrasing John Stuart Mill (the Education Committee quotes him to some effect), we may say that a doctor who is only a doctor is not even a good doctor. In spite of our special interest in medical schools, we may still focus our attention on universities as a whole. If universities are overcrowded and an effort to bring about a more satisfactory state of affairs is to be made, one of several things must be done—universities must grow in number or in size, or fewer students must be granted admission to them. Though a very good case may be made out for the curtailment of the number of students allowed to enrol every year as starting on their medical careers and some consideration will have to be given to this possibility before very long, it is doubtful whether anything will be done, or even should be done, to place obstacles in the way of any student who seeks entrance to a university. This freedom of entry can be tolerated only if universities maintain their traditional method in which education in the true sense is the first consideration. In the present discussion all the ground cannot be covered and no detailed reference can therefore be made to the degree of preparation that a student should undergo at his secondary school before he is enrolled as an undergraduate. If universities are to expand either in size or in number, money, much money, will be needed. This will have to be provided by the State. It seems that no real sense of values can be instilled into the minds of State authorities. If this was possible we should not hear the recurrent cries for help at the start of the academic year by an institution that lives on the good-will of its politicians; nor should we see newspapers publishing leading articles on the "financial crisis" faced by the university. As the Nuffield Subcommittee reminds us, money alone will not suffice. Money will accelerate expansion, but by itself it will not produce the teachers and research workers on whom everything else depends. "If universities are to expand rapidly they must have priority in the allocation of building labour and materials for laboratories, lecture rooms, residences for the additional staff, and residential accommodation for graduate and undergraduate students. They need paper for texts and text-books; furniture and equipment; priorities in general to make the additional money grants effective." In the matter of money it is interesting to read that the whole university system in Great Britain costs £10,000,000 a year; that £104,000,000 are spent on books, magazines and newspapers; that tobacco cost £603,000,000 and alcoholic beverages £680,000,000. One can imagine with what joy the public would receive the suggestion that a tax on cigarettes and beer should be used to further university education! One warning of the utmost importance is made by the subcommittee, and that is the danger of the worship of size. To state this danger should be sufficient, for when size is set up as something to be admired, quality of education must inevitably suffer. There are really two problems—a short-term and a long-term problem. For the former our subcommittee can find no satisfactory answer. In regard to the long-range problem the creation of new universities is mentioned. It is stated that a wider geographical distribution of universities is needed. This need has been stated by this journal for Australia. We are warned that the creation of new universities in new areas will create an additional demand of its own. In all this planning government funds will be needed, and if university development is to be satisfactory governments will have to take care not to interfere in any way that will be a hindrance. The subcommittee states that the drive for uniformity which so often follows an extension of State activity is due very largely to considerations of administrative convenience and economy which are not very relevant or important in the field of university development. Some persons would describe this statement as wishful thinking, but most will agree that it is probably correct. Most of those who have given this whole subject serious thought will agree with the subcommittee that a case may be made for leaving the universities their present freedom to expand or contract along the lines suggested to them by their conceptions of what they can best do with their resources and of how they can contribute most to

local and national needs. On this note we would end the present discussion, emphasizing once again the essential over-all requirement that education on broad lines, and not technical instruction, must always remain the aim of universities.

Current Comment.

SUDDEN DEATH OF BABIES.

FEW things are more shocking to the general public, and especially to parents, than the sudden death of an apparently healthy baby. Particularly disturbing to conscientious parents is the type of death recently reported, and occurring from time to time, in which the child has been found face downwards in the cot or bassinet and the coroner has brought in a verdict of death by asphyxiation, the child apparently having smothered itself accidentally with its own pillow. Another type of death appears to be due to the inhalation of food particles particularly when vomiting is associated with crying. Last year an anonymous article appeared in a well-known American magazine apparently written by the father of a boy, aged ten months, who was found dead in his crib. The story was that the baby had an area of irritation on the back of his head and the doctor advised the mother to put the baby face downward during his nap. This the mother did. The baby was heard to cry and vomited a part of his previous meal; when the parents reached the bed he was dead. The father felt that the death was due to the fact that the child was placed in the prone position on the doctor's advice and was apparently very critical of the medical profession for giving such advice. The question was referred by a doctor to the "queries" column of *The Journal of the American Medical Association* for an opinion. In the reply[1] it was stated that the evidence was insufficient to prove that the prone position of itself was the cause of sudden death. Anyone who has observed the sleeping habits of babies, as in a hospital ward, will know that many sleep in the prone position by choice, with no apparent ill effects. The reply to the query stresses, however, the importance of a firm mattress completely filling the cot, so that the child may not bury his face in it or become wedged between it and the side of the cot; pillows are proscribed completely. In an analysis of deaths of infants recorded at the Leeds coroner's court, C. J. Polson and D. E. Price[2] state that soft feather pillows were the factors responsible in two deaths and probably those responsible in another two. It is not quite clear what they mean, but as their discussion refers primarily to deaths associated with vomiting, it is doubtful that they imply the occurrence of suffocation by the pillows alone. They stress the importance of careful preparation of artificial milk foods to ensure that lumps are avoided and refer unfavourably to the practice of enlarging the opening of a teat so that these lumps are sucked through. The American discussion also refers to asphyxia due to the aspiration of food particles, including milk curds and mucus. It seems logical enough that an infant face-down on a soft pillow or mattress will more readily aspirate vomitus than if it has free means of escape, and the wisdom of the firm mattress and pillow (if any) is apparent. Australian authorities recommend to mothers the use of a fibre mattress with a light chaff mattress on top and a small chaff pillow, and this would seem to meet the situation. The question of death apart from vomiting remains vexed. The reply in the American journal lists, among reported or conjectured causes of sudden death, accidental strangulation with bands of clothing, blankets and restraining garments, acute bronchopneumonia, spontaneous hæmorrhages into the lungs or peritoneal cavity, apoplexy of both adrenal glands, hyperacute attacks of fever as in meningococcus infections, anaphylactic shock, laryngospasm of tetany, and hyperglycæmia due to adrenal insufficiency, and accepts the occurrence of simple self-asphyxiation with a pillow while rejecting the once popular "thymic death". The problem is by no means fully resolved; there is room for careful investigation and need for the development of prophylactic measures.

[1] *The Journal of the American Medical Association*, May 29, 1948, page 495.
[2] *The Lancet*, February 28, 1948.

THE INDEX-CATALOGUE OF THE LIBRARY OF THE SURGEON-GENERAL'S OFFICE IN AMERICA.

THE Army Medical Library in Washington, D.C., has become known all over the world through being the home of the Index-Catalogue. The Index-Catalogue has a fundamental role in medical research as a subject-bibliography and is the basis of medical library service in the United States and abroad. A library of such huge proportions as the Army Medical Library would remain just a museum or storehouse of books and journals if there were no proper key to the treasures of past and present medical experience hidden in millions of printed pages. The ordinary card catalogue of the library can take care only of the titles of books and journals, without revealing anything of what, for instance, a 50-volume set of a medical serial may contain. An indexing-journal or list also is unable to give more than scanty information on the current topics of medical literature, and does not penetrate beyond the surface of contemporary research. The card catalogue is just a local tool, and the Current List, or any indexing-journal, is just an improvised first aid measure.

Medical research needs a complete exhibition of the contents of its progress, a full view of the material that has accumulated throughout the centuries; it needs an institution where the scientific literary resources of medicine are analysed, sieved, classified, and made public for the benefit of not one library, or one research laboratory, but of all medical men of the world. Such an institution is the Index-Catalogue established by an act of the Congress of the United States in 1879. The Index-Catalogue is the medico-bibliographical survey carried out uninterruptedly by the United States Government. The material of this survey is what the Army Medical Library holds in books, pamphlets, and serial publications. The more complete the stacks of the Library become, the closer will the bibliographical survey approach the ideal point of exhaustiveness. Everything received by the Library, whether old or new, whether printed, written, or mimeographed, is analysed, examined and recorded with a view to its present or future usefulness for medical research. The result of this analytical work of the Index-Catalogue is a file of 1,500,000 cards, arranged in one alphabet of many thousand medical subjects: the "manuscript" Index-Catalogue, ready to serve local visitors as well as scholars in remote places.

The second part of the work of the Index-Catalogue as an institution is the synthesis and printing of the medico-bibliographical encyclopædia. This is the printed Index-Catalogue or the "Surgeon-General" as it has been called by British librarians since 1880. As a large encyclopædia, it has many volumes in its complete alphabetical set, and since the growth of medical literature is continuous, it has several alphabetical sets. The present set is the fourth, and it has proceeded as far as the first half of the letter M, which has just been published. This is a matter for congratulation. Altogether the printed Index Catalogue comprises 57 volumes thus far, and lists 3,357,755 references, 2,865,201 of which are arranged under subjects. The number of literary sources revealed by both the printed and the manuscript Index-Catalogue amounts to almost 5,000,000—all of them available in the Army Medical Library.

The printed Index-Catalogue is found in public and medical libraries in many countries; it is available in many medical libraries in Australia. It is the only comprehensive subject-bibliography of medicine and allied sciences, called by Welch the greatest gift the American people and American medicine give to mankind.

Abstracts from Medical Literature.

GYNÆCOLOGY.

Adenomyosis of the Uterus with Tuberculous Infection.

R. DE SOLDENHOFF (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1948) reports a case of adenomyosis of the uterus with associated tuberculous infection, and reviews the literature on the subject; he states that only 25 cases have been reported. In this case a nulliparous patient, aged thirty-eight years, had uterine hæmorrhage of fourteen days' duration and some lower abdominal pain. A diagnosis of tuberculous endometritis had been made previously on biopsy after curettage, but there was no evidence of pulmonary or urinary tuberculosis. At operation a large mass filled the lower abdomen to the level of the umbilicus. Hysterectomy was performed. It was difficult to find the peritoneal cavity on account of adhesions between bowel and the uterine mass and parietal peritoneum. The mass consisted of a non-capsulated tumour occupying most of the fundus of the uterus with a cavity filled with blood clot and necrotic material. It was typically adenomyomatous with tuberculous giant cells grouped around the glandular elements. The uterine muscle did not appear to have been infected. It would appear that adenomyosis was originally present and afforded an extra area of hyperplastic glandular epithelium offering a fertile field for tuberculous invasion. The author states that deep X-ray therapy, sanatorium treatment and intraperitoneal instillation of oxygen have been used in the treatment of tuberculous infection of the female genital organs, but conservative treatment was out of the question in the case described. He considers that total hysterectomy is to be preferred to the subtotal operation and makes a plea for radical operative treatment of tuberculosis of the uterus and adnexa.

The Technique of Vaginal Hysterectomy.

N. SPROAT HEANEY (*The Western Journal of Surgery, Obstetrics and Gynecology*, July, 1948) describes some changes in his technique of vaginal hysterectomy. He states that the appropriate instruments, two good spot lights and two trained assistants are indispensable to the best technique. Pre-operative preparation of the patient is important, and the anæsthetic of choice is "Sodium Pentothal" or ethylene gas after premedication by "Demerol" or barbiturates. The abdomen is always prepared for possible laparotomy. The cervical os is closed by sutures if infection or malignant disease of the endometrium is suspected. If the cervix is infected it is first cauterized. If stress incontinence is present the dissection of the vaginal mucosa is taken up to the urethral meatus and urethroplasty is performed before the uterus is removed. The anterior cul-de-sac is first opened and then the posterior cul-de-sac. Most of the bleeding in a vaginal hysterectomy comes from the posterior colpotomy incision. The sacro-uterine ligaments are cut and tied, the uterus is drawn downwards, and clamping, cutting and ligating with figure-of-eight chromicized sutures is continued along each side of the uterus. If the uterus is too large to be delivered intact the cervix is amputated to facilitate delivery of the body. A large tumour is removed by morcellation. If after the uterine vessels have been tied it is found impossible or unwise to proceed further by the vaginal route, the cervix may be amputated, the vaginal vault closed and the rest of the operation performed abdominally. Hæmostasis must be complete before reconstruction of the pelvic floor is commenced. In cases of prolapse the round ligaments are attached to the anterior vaginal wall just beneath the urethra and the broad ligaments are sewn together in the midline from top to bottom. The sacro-uterine ligaments are approximated as high as possible inside the pelvis. A posterior repair (if necessary) completes the operation. The author never isolates the levators from their fascial beds, nor does he recommend suturing them as a separate layer. Catheterization is carried out at six-hourly intervals until the patient passes urine spontaneously and the residual urine is not more than one ounce. The patient is urged to sit on the edge of the bed on the day after operation, and goes to the toilet with help the following day. Usually the patient can walk freely on the fifth day and leaves hospital on the seventh post-operative day.

Pneumo-Pyosalpinx.

R. W. KINROSS PURSER AND JAMES YOUNG (*The Journal of Obstetrics and Gynæcology of the British Empire*, August, 1948) report an unusual case of acute pneumo-pyosalpinx and discuss the possible causes of gas in the cystic swelling. The patient was a nulliparous woman, aged thirty-two years, with a history of severe low abdominal pain of sudden onset and twenty-four hours' duration. The symptoms and signs pointed to a right-sided tubal infection, but there was resonance over the tumour where dulness was to be expected. Two years previously the patient had undergone appendicetomy and drainage of an abscess associated with a retrocaecal appendicitis. The menstrual history was normal, and pelvic examination revealed fullness and acute tenderness through the right fornix. The patient was treated with penicillin and "Sulphatriad" with prompt improvement in her general condition, but an evening rise in temperature persisted and the mass failed to diminish in size. Examination under anæsthesia was carried out on the tenth day, and after the aspiration of pus through the posterior fornix and posterior colpotomy, laparotomy was performed. A cystic tumour, six inches in diameter, arising from the right tube and ovary was freed from bowel and omentum and removed. This cyst contained two-thirds gas and one-third pus, and culture of pus from the cyst yielded a moderate growth of coliform bacilli. Recovery was satisfactory except for a sinus in the lower end of the abdominal wound, which later healed. X-ray examination showed no evidence of bowel fistula and appropiate tests showed no evidence of diabetes. It was apparent that the patient had a pyosalpinx which had undergone sudden enlargement following reinfection and introduction of gas. Cultures showed no evidence of organisms of the gas gangrene group, but gas-forming coliform bacilli were the chief organisms grown from the cyst and from the discharging sinus. Under certain circumstances coliform bacilli produce more gas than usual, particularly in a suitable sugar medium and in diabetics. The authors state that the bulk of evidence in this case suggests a small fistulous communication between the cyst and bowel. Salpingo-colic fistula complicating a pyosalpinx has been described and a fistulous communication between the small bowel and a pyosalpinx is quite possible. A small fistula may have permitted the entrance of gas and organisms into the cyst, although no such fistula was demonstrated either at operation or radiographically.

Relief of Severe Stress Incontinence.

C. MCINTOSH MARSHALL (*The Journal of Obstetrics and Gynæcology of the British Empire*, April, 1948) discusses suprapubic fascial sling operations for the relief of severe stress incontinence and describes the technique of two such operations which he has employed with success. The author considers that the Aldridge operation is unnecessarily complicated, the vaginal portion of the operation being not necessary for patients with stress incontinence in whom there is no urethrocele, cystocele or combination of both; the urethra can be located and encircled from above with as much ease as by the usual vaginal approach. The general opinion that sling operations should be used only when other so-called simpler methods have failed is unjustified, and the operation should be used as a primary effort in selected cases. The operations described are neither difficult nor fraught with dangerous complications. For patients with prolapse and slight stress incontinence a Manchester type of operation is performed; for patients with prolapse and pronounced stress incontinence a sling operation is added to the vaginal procedure. Patients who have had previous unsuccessful vaginal operations and those with no associated prolapse are treated by the sling operation performed by the suprapubic and retropubic route. The operation is a suprapubic procedure to sling the urethra with two strips of fascia raised from the anterior parts of the rectus sheaths and pedicled laterally in the external and internal oblique muscles.

Bacteriology of the Vagina.

F. R. LOCK, M. D. YOW, M. I. GRIFFITH AND C. STOUT (*Surgery, Gynecology and Obstetrics*, October, 1948) report a study of the organisms in the vaginas of 75 normal adult women and briefly review the literature on the subject. The patients investigated were student nurses, between the ages of seventeen and twenty-five years, with no gynæcological symptoms. All techniques for obtaining vaginal cultures are stated to be unsatisfactory, but apparently satisfactory results were obtained by the use of cotton swabs in glass tubing which were pushed out of the protective tubing after the vaginal cavity had been entered. The pH of the vaginal secretion was noted in each case, nitrazine paper being used. In ten cases no organisms were grown, in 37 lactobacilli only, in 25 lactobacilli and other organisms, and in three organisms other than lactobacilli. The authors point out that the term

Döderlein's bacillus is being replaced in recent books by *Lactobacillus acidophilus*. They state that study of direct smears for organisms other than lactobacilli are of little value, but when these are combined with suitable culture procedures there is presumptive evidence that lactobacilli are present in 100% of cases. Streptococci were present in 18% of the cultures and the anaerobic variety predominated. There was an associated growth of lactobacilli in thirteen of the fourteen cases in which streptococcal cultures were grown. Lactobacilli are best cultured in an atmosphere of 10% to 20% carbon dioxide on chocolate agar plates. There is no direct correlation between the presence of lactobacilli in the vagina and its degree of acidity. Healthy unmarried young adult females usually harbour lactobacilli within the vagina; about one-half also harbour other organisms, chiefly diphtheroids, *Staphylococcus albus* and streptococci. Vaginal streptococci are adapted to an environment of low oxygen content.

OBSTETRICS.

Lipiodol Tubal Insufflations for Sterility.

R. N. RUTHERFORD (*The Western Journal of Surgery, Obstetrics and Gynecology*, March, 1948) records the results among 417 patients with either primary or secondary sterility due to tubal occlusion who were treated by repeated lipiodol insufflations. All patients were thoroughly investigated before treatment; they were proved to be habitually ovulating, and tubal occlusion was diagnosed after two failures of air insufflation. Twelve millilitres of lipiodol were used at each insufflation and the subjective reactions of the patient were found to be a good index of the distending pressure. In the presence of tubal occlusion the lipiodol injected caused an increasing pressure on the utero-tubal junctions and on the tip of the cannula and afforded the effective dilating pressure for the tubes. Two X-ray pictures were taken, the first immediately after the twelve millilitres had been injected, and the second ten minutes later. Such utero-tubograms were prepared once monthly until tubal patency was established; after this insufflation was repeated at three-monthly intervals until pregnancy ensued. After the failure of three consecutive efforts to make the tubes patent, surgery was offered or child adoption was suggested. The authors state that the selection of the fertile phase for carrying out the procedure is most important. In the series of patients investigated the application of monthly lipiodol insufflations relieved proved tubal occlusion in 71% and subsequently pregnancy occurred in 63% of cases. Amongst 264 women who conceived, the fœtus was viable in 233 cases and spontaneous abortion occurred in 31. The method failed to relieve tubal occlusion in eleven patients, who were treated surgically, and three of these became pregnant subsequently. There was one death amongst the patients treated; this was due to acute generalized gonococcal peritonitis. Pelvic inflammation followed as a complication in 2·4% of cases. Oil was found by X-ray evidence to have been injected into the uterine venous sinuses of two patients, but they showed no untoward effects of oil embolism. On an average 2·8 utero-tubograms were required to effect patency for each patient. Pregnancy occurred despite failure to prove tubal patency in eleven cases. Perseverance on the part of the patient and the physician is of importance in such a method of treatment.

A Follow-Up Study of Eclampsia.

LEON C. CHESBY, W. H. SIEMERS AND F. H. VANN (*American Journal of Obstetrics and Gynecology*, September, 1948) review 245 cases of convulsive and three cases of non-convulsive eclampsia which have occurred at the Margaret Hague Maternity Hospital. The incidence was 0·3%, the immediate mortality (maternal) 10% and fœtal loss 33%. Since 1945 the greater number of the survivors have been followed for at least a year or until death which has occurred in twelve cases—six in the cardio-vascular and renal field. The remote annual death rate among the immediate survivors of eclampsia has been 6·390 per 1000 or two to four times the expected rate. The incidence of hypertension (which was assessed on a systolic blood pressure of 170 millimetres of mercury or more and a diastolic pressure of 90 millimetres or more) was 15% and a further 7% had an increase in the systolic or diastolic pressure alone. There was a reduced incidence of hypertension in patients affected over the past six years compared with the earlier patients; the authors suggest that this is the result of the more radical management of the disease. There were 226 pregnancies subsequent to eclampsia; the gross fœtal loss was 46 or 20·3%. The recurrence rate amongst pregnancies carried on to the stage of fœtal viability was 34%, and 45% of the women pregnant again had at least one toxæmia. The unfavourable factors affecting the prognosis in regard to future pregnancies are defined as follows by the authors: (i) an initial systolic blood pressure above 120 millimetres of mercury, (ii) an average blood pressure in the eclampsia above 160 millimetres of mercury, (iii) duration of toxæmia of more than one week, (iv) failure of the blood pressure and urine to return to normal by the tenth post-partum day, (v) a weight/height ratio, at follow-up, greater than 2·2 pounds per inch.

The Prognosis of the Cardiac Patient in Pregnancy.

STANLEY LESSE (*American Journal of Obstetrics and Gynecology*, September, 1948) summarizes the cardiac abnormalities which contraindicate a contemplated pregnancy as follows: (i) pronounced cardiac decompensation during a previous pregnancy or evidence of moderate to severe cardiac failure during the non-pregnant period, (ii) much cardiac enlargement, (iii) mitral stenosis with incidence of myocardial failure, (iv) syphilitic aortic regurgitation, (v) persistent auricular fibrillation, (vi) subacute bacterial endocarditis, (vii) the presence of acute rheumatic fever, (viii) certain types of congenital heart disease. A study by the author of 203 pregnant cardiac patients, representing 3·2% of 6285 conservative pregnancies, showed that 54 (or 26·6%) suffered from decompensation at one time or another during the course of the pregnancy. Of patients who gave a history of moderate to severe decompensation either during a previous pregnancy or during the non-pregnant period, 71% developed definite signs of cardiac failure, although there was no evidence of cardiac failure at the time of the first prenatal visit. Thirty-two of 153 patients (20·9%) who were thirty years old or younger suffered from decompensation, while 22 of 50 patients over the age of thirty years suffered from decompensation. This indicated that, if women known to have cardiac disease desire to have children, their pregnoncies, if permitted at all, should take place during the early years of married life. For women of the same age, parity had little or no effect on prognosis unless there was a previous history of decompensation. The mitral stenotic murmur was by far the most common auscultatory abnormality affecting the prognosis of the pregnant cardiac patient. In the series 55·9% of patients in classes (ii), (iii) and (iv) having mitral stenosis suffered from decompensation. Of the 54 cases of decompensation, 47 occurred during the course of pregnancy before labour began, one during the course of labour, and six in the puerperium within a period of several days after delivery.

The Outcome of Pregnancy following Vaginal Operations.

WILLIAM F. FINN (*American Journal of Obstetrics and Gynecology*, August, 1948) draws attention to the divergent opinions on the effects of vaginal operations on subsequent pregnancy, and formulates general principles for the treatment of vaginal lesions during the reproductive years. A study was made of 390 patients who had one or more deliveries after a vaginal operation. In this series, 188 patients had minor vaginal operations and no ill effects were noted in subsequent labour, nor did labour have any effect on the operative results. Among 202 patients who had major vaginal operations the incidence of subsequent abortion or premature labour was not higher than for average women. The cervix dilated without obvious difficulty, and cervical lacerations were not more extensive or more frequent than was customary. Descent of the head was delayed by the vaginal scars in 3·5% of cases, and deep mid-line episiotomies were occasionally necessary to facilitate delivery. There was a striking increase in the incidence of Cæsarean section, which was performed in 8·5% of cases, this being more than twice the clinic average of 3%. There were only two recurrences amongst fifteen patients who had vaginal delivery following repair of vaginal fistulæ. The Manchester operation had comparatively minor effects on the course of labour, but recurrence of prolapse was noted in four cases. The majority of patients who had surgical treatment for vaginal fistulæ, third degree lacerations and vaginal plastic operations delivered themselves vaginally without too many untoward results. The author concludes that it is not necessary to urge that childbearing be completed prior to vaginal operations. There is no indication to interrupt pregnancy occurring after vaginal operations. In certain instances the danger of injuring extensively repaired vaginal tissues should be obviated by Cæsarean section. In a few cases the patient must be warned that recurrence of the vaginal lesion after childbirth may necessitate a secondary operation.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the Queensland Branch of the British Medical Association was held at B.M.A. House, Wickham Terrace, Brisbane, on December 10, 1948, DR. NORMAN SHERWOOD, the President, in the chair.

ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council which had previously been circulated among members was taken as read and adopted on the motion of Dr. T. V. Stubbs-Brown, seconded by Dr. Harold Love. The report is as follows.

The Council has pleasure in presenting the following report of the work of the Branch for the year ending November 15, 1948.

Membership.

The membership of the Branch is 732, *plus* 4 honorary members and 1 complimentary member, as against 715, and 5 honorary members and 1 complimentary member in 1947, making a total gain of 17. There are also 128 honorary associate members, 21 of whom were elected this year.

The gains were: new members 29, transfers from other Branches 25, members reinstated 3.

The losses were: members transferred from Branch 29, resignations 1, deceased 5, struck off 5 (including one member whose address is unknown).

Obituary.

We regret to record the deaths of the following members: Dr. A. Jefferis Turner, Brisbane; Dr. Paul Voss, Rockhampton; Dr. D. Gifford Croll, Brisbane; Dr. O. B. Goyen, Beaudesert; Dr. H. P. Blaney, Howard.

David Gifford Croll.

A great loss has been suffered by the Branch in the death of Dr. Gifford Croll, which took place on May 4, 1948. Dr. Croll was a keen and active member of the Council for many years and held the office of President in 1924. He represented the Branch on the Federal Committee and later on the Federal Council, when that body was established, and occupied the position until he went on service during the second world war. He was also a director of the Australasian Medical Publishing Company, Limited, from 1929.

Although he was no longer a councillor at the time of his death he was still a member of several standing committees.

In 1946 he was elected a vice-president of the Branch in recognition of the valuable services given by him during the past twenty-seven years.

Alfred Jefferis Turner.

On December 29, 1947, the death of Dr. A. J. Turner occurred at the age of eighty-six years. Dr. Turner was a noted member of the Branch and a former president.

Meetings.

In addition to the annual meeting, ten general meetings of the Branch were held, including two clinical meetings; also a special general meeting was called to discuss further information with regard to *The Pharmaceutical Benefits Act*, 1947. The average atendance at the ordinary meetings was 46, and 178 members attended the special general meeting and seven visitors.

Council.

Twenty-three Council meetings were held. Record of attendance is as follows:

Dr. Norman Sherwood (President)	23
Dr. B. L. W. Clarke (President-Elect)	17
Dr. H. W. Johnson (Past President)	17
Dr. H. W. Horn (Honorary Treasurer, Federal Council Representative, Chairman of Committees)	20
Dr. Arnold Robertson (Honorary Secretary)	22
Dr. J. R. Adam (Honorary Secretary of Committees)	22
Dr. R. V. Adamson (Councillor)	14
Dr. F. W. Arden (Councillor)	15
Dr. T. V. Stubbs Brown (Councillor)	19
Dr. K. B. Fraser (Councillor)	17
Dr. Milton Geaney (Councillor)	18
Dr. Glen. V. Hickey (Councillor)	19
Dr. Alan E. Lee (Councillor, Federal Council Representative)	19
Dr. Harold R. Love (Councillor)	19
Dr. F. W. R. Lukin (Councillor)	17
Dr. Athol Quayle (Councillor)—on leave from May 14, 1948)	9
Dr. R. G. Quinn (Councillor)	16
Dr. W. H. Steel (Councillor)	19

Scientific and Medico-Political.

February.—Clinical meeting in conjunction with the Brisbane Hospital Clinical Society.

March.—Dr. D. P. H. Schafer: "Some Aspects of a Recent Medical Refresher Course in England."

April.—Dr. J. H. Willson: "Clinical Applications of Recently Introduced Methods in General Anæsthesia."

May.—Dr. R. B. Salter: "The Diagnosis and Management of Placenta Prævia."

May.—Special General Meeting: *"The Pharmaceutical Benefits Act*, 1947."

June.—Dr. E. H. Derrick: "The Spirit of the Researcher." (The Bancroft Oration.)

July.—Dr. H. R. Love, Dr. D. C. Jackson and Dr. Alan E. Lee: "The Psychosomatic Approach in Medicine."

August.—Dr. S. C. Suggit: "The Indications for Tonsillectomy."

September.—Dr. H. S. McLelland: "Ancient and Modern Medicine Men." (Jackson Lecture.)

October.—Mr. A. H. Penington: "The Active Treatment of Pulmonary Tuberculosis."

November.—Clinical meeting in conjunction with the Mater Misericordiæ Hospital Clinical Society.

Joseph Bancroft Oration.

Dr. E. H. Derrick delivered the twenty-third Bancroft Oration on Friday, June 4, 1948, in the Medical School lecture hall. He chose for his subject "The Spirit of the Researcher".

This is the first occasion on which the Bancroft Oration has been delivered by a member of the Branch and the high standard set by previous orators was well maintained by Dr. Derrick.

The oration was well attended by members and visitors. The President expressed gratification at the presence of Dr. Josephine Mackerras, who is a granddaughter of Joseph Bancroft.

A vote of thanks was moved by Dr. Ian Mackerras and seconded by Professor H. J. Wilkinson.

At the conclusion of the oration the Bancroft Medal was presented to Dr. Derrick.

Jackson Lecture.

On September 3 the eighteenth Jackson Lecture in memory of Ernest Sandford Jackson was given by Dr. H. S. McLelland on the subject of "Ancient and Modern Medicine Men". The lecture was most interesting and at the conclusion a vote of thanks was moved by Dr. F. W. R. Lukin, seconded by Dr. Robin Charlton and carried by acclamation.

Office Bearers and Councillors.

Dr. B. L. W. Clarke was elected President-Elect for the year 1948.

Honorary Secretary.—Dr. Arnold W. Robertson was elected to the position of Honorary Secretary, but is not standing for reelection. The Council wishes to record its appreciation and thanks for the capable manner in which he has carried out his duties.

Council.—No changes have taken place in the personnel since the election. Dr. Athol Quayle, who went overseas, was granted leave of absence.

A number of councillors are not seeking reelection for the ensuing year: Dr. R. V. Adamson, Dr. T. V. Stubbs Brown, Dr. Milton Geaney, Dr. Horace Johnson, Dr. F. W. R. Lukin and Dr. W. H. Steel. Most of them have given many years of valuable service to the Branch and it is hoped that their example will be followed by other members who are prepared to sacrifice their time and energy to the cause of the profession.

The following office bearers were elected by the Council:

Honorary Treasurer.—Dr. H. W. Horn (reelected).
Chairman of Committees.—Dr. H. W. Horn (reelected).
Honorary Secretary of Committees.—Dr. J. R. Adam.
Honorary Librarian.—Dr. Neville Sutton (reelected).
Assistant Honorary Librarian.—Dr. Konrad Hirschfeld (reelected).

For a number of years an open invitation has been extended to members of the Branch to attend Council meetings as observers. Councils of Branches in all States have issued an invitation to members of the Queensland Branch Council to attend their meetings as observers should they be visiting the State at any time.

Ethics Committee.

At the annual meeting of the Branch held on December 12, 1947, the following were elected members of the Ethics Committee: Dr. Graham Sutton, Dr. Val McDowall, Dr. J. J. Power, Dr. Gifford Croll, Dr. J. G. Avery, Dr. L. J. J. Nye, Dr. J. G. Wagner. The ex-officio members of the Ethics Committee consist of the president, president-elect, the honorary secretary and the honorary treasurer for the time being in office. No meetings were held during the year.

Library.

During the year 125 books were borrowed from the library by 40 members. Arrangements have been made for country members to borrow medical books and bound journals from the Branch library on condition that they defray cost of postage. A list of medical periodicals dealing with medicine and allied sciences, in the technical libraries of Brisbane, including the Branch library, drawn up by the Queensland Institute of Medical Research, was sent to local associations early in the year, and members were advised that a copy of the list would be sent to individuals on application to the Secretary of the Post-Graduate Medical Education Committee.

The following journals have been added to the library during the year: *The Journal of the Canadian Medical Association, Tubercle, The American Journal of Surgery,* "Harvey Lectures, 1945-1946", *The Journal of Bone and Joint Surgery, The British Journal of Urology, Archives of Pediatrics, The Laryngoscope, La presse médicale, The British Journal of Anæsthesia, The British Journal of Tuberculosis, Archives of Otolaryngology, Postgraduate Medical Journal, Nature, Swiss Medical Journal, South African Medical Journal,* and the following yearbooks, 1946: "General Surgery", "Radiology", "Industrial and Orthopedic Surgery", "Obstetrics and Gynecology", "Urology", "Ear, Nose and Throat", "Dermatology and Syphilology", "Endocrinology and Nutrition", "General Therapeutics", "Physical Medicine", "Pediatrics", "General Medicine".

Also "British Surgical Practice", the first three volumes of which have been delivered.

The following have been donated to the library: by Mrs. A. Jefferis Turner, her late husband's collected papers, and by Dr. J. G. Avery, "Nelson's New Loose-leaf Surgery".

Representation.

The Branch was represented as follows during the year:

Council of the British Medical Association.—Dr. Isaac Jones.

British Medical Association Representative Meeting, Cambridge, 1948.—Dr. H. M. Whyte.

Federal Council of the British Medical Association in Australia.—Dr. H. W. Horn, Dr. A. E. Lee.

Federal Council Contract Practice Committee.—Dr. L. P. Winterbotham.

Australasian Medical Publishing Company, Limited.—Dr. D. Gifford Croll (director), Dr. Alan E. Lee (director), Dr. T. A. Price, Dr. H. W. Horn (members).

Medical Assessment Tribunal.—Dr. Alec Marks.

Queensland Medical Board.—Dr. D. Gifford Croll, Dr. R. G. Quinn, Dr. J. G. Wagner, Dr. F. W. R. Lukin.

Medical Officers' Relief Fund (Federal).—Queensland Committee: Dr. W. H. Steel, Dr. G. W. Macartney, Dr. K. B. Fraser.

Post-Graduate Medical Education Committee.—Dr. A. E. Lee, Dr. Felix Arden, Dr. Harold R. Love.

Queensland Institute of Medical Research.—Dr. W. H. Steel.

Queensland Bush Nursing Association.—Dr. L. Bedford Elwell.

Queensland Council of Social Agencies: Social Studies.—Dr. G. B. V. Murphy.

Flying Doctor Service of Australia.—Dr. Harold Crawford.

Red Cross Blood Transfusion Service.—Dr. Milton Geaney.

The Surf Life Saving Association of Australia (Queensland State Centre).—Dr. F. W. R. Lukin (Liaison Officer).

National Safety Council of Australia (Queensland Division).—Dr. L. A. Little.

Physical Fitness Association of Queensland.—Dr. E. S. Meyers, Dr. Harold Crawford.

Australian Association for Better Hearing.—Dr. Herbert Earnshaw, Dr. J. R. Hutcheon.

Queensland Bush Children's Health Scheme.—Dr. Felix Arden.

Board of Studies in Physiotherapy.—Mr. J. R. S. Lahz.

Federal Medical War Relief Fund: Local Committee of Management.—Dr. J. G. Wagner, Dr. F. W. R. Lukin, Dr. M. Geaney, Dr. J. G. Avery, Dr. J. V. Duhig.

Queensland Health Education Council.—Dr. Felix Arden.

British Commonwealth Medical Council (Inaugural Meeting).—Dr. Athol Quayle.

Florence Nightingale Memorial Committee of Australia (Queensland Branch).—Dr. Norman Sherwood.

The Editor of THE MEDICAL JOURNAL OF AUSTRALIA was represented by Dr. Felix Arden.

Organization Subcommittee.

Personnel.—Dr. H. W. Horn (chairman), Dr. J. R. Adam, Dr. F. Arden, Dr. K. B. Fraser, Dr. F. W. R. Lukin, Dr. Athol Quayle, Dr. R. G. Quinn, Dr. T. V. Stubbs Brown, and the ex-officio members of the Council. Twenty-three meetings of the subcommittee were held at which various matters were dealt with, or recommendations submitted to the Council where policy was involved. This subcommittee is of great assistance to the Council in its work.

By-Laws.

Amendment of By-Law 4: Members' Subscriptions.—Notice has been given to members in accordance with Article 67 (2) of the proposed amendment of By-Law 4 to increase subscription rates, to be submitted to the annual meeting of the Branch to be held on December 10, 1948. The Council considers the proposed amendment not only justified, but also unavoidable. Branch expenses have increased considerably owing to publicity necessitated by *The Pharmaceutical Benefits Act* and the subscription rate payable to the Federal Council has been raised from 10s. (1947) to 15s. (1948), and in 1949 the amount will be 21s. per member. When the full-time secretariat of the Federal Council is established there will be a still further increase.

Reelection of Council: By-Law 8.—The method of election of councillors was discussed at the annual conference of local associations held in June last and the question has been submitted to the Branch solicitors for an opinion.

Advertisement (By-Law 22).—The opinion was expressed by the Council in regard to newspaper publicity that the responsibility rests with the members to see that the by-laws of the Branch are adhered to in regard to any statements given to the Press.

Lodges.

Although the question of stabilization of the lodge capitation fee in Queensland has not been finalized, many country districts have adopted the sliding scale principle in accordance with the rise and fall of the Nominal Wage Index figure, which has been in operation in the metropolitan area for a number of years. The rate announced by the Government Statistician's office for the current year, to operate from July 1, 1948, was 37s.

Conferences with representatives of the Queensland Friendly Societies Association regarding the question of capitation fee and income limit took place. The Council has given consideration to fluctuation in income limit in accordance with the rise and fall in the Nominal Wage Index figure on parallel lines with the method of fixation of the capitation fee. In reply to an inquiry received from the Federal Council the Queensland Branch Council expressed disagreement with the abolition of the upper income limit.

Building Subcommittee.

Personnel.—Dr. M. Graham Sutton, Dr. R. G. Quinn and Dr. K. B. Fraser. Two meetings of the subcommittee were held: (i) to deal with the finalizing of the purchase of the property in Bartley Street at the rear of B.M.A. House; (ii) to go into the matter of electrical installation in B.M.A. House.

Liquid Fuel Subcommittee.

Personnel.—Dr. L. P. Winterbotham, chairman, Dr. K. B. Fraser, Mr. F. K. Davis, secretary. The whole of the fuel ration of city members was rescinded by the authorities, and the committee was instrumental in securing adequate supplies for members. In this work the subcommittee was assisted by Dr. A. E. Paterson, a former member.

Monthly Newsletters.

The Publicity Committee is still continuing to produce an interesting newsletter each month which is greatly appreciated by members of the Branch.

Memorial Roll, Queensland Branch of the British Medical Association.

Four names have been added to the Branch Memorial Roll during the year, namely, Sydney Fancourt McDonald, Graham Patrick Dixon, Leonard Hardwick Foote and Alfred Jefferis Turner.

Medical Fees Tribunal.

Personnel.—Dr. J. G. Wagner (chairman), Dr. Alan E. Lee (honorary secretary), Dr. R. G. Quinn, Dr. G. W. Macartney, Dr. H. S. McLelland, Dr. Norman Sherwood. A tribute to the late Dr. D. Gifford Croll, who was a member of the tribunal from its inception in March, 1939, was placed on record. Dr. Norman Sherwood was appointed to fill the vacancy caused by the death of Dr. D. Gifford Croll.

Four meetings were held at which seven cases were dealt with, six of which were referred by the Insurance Commissioner concerning workers' compensation medical fees. Two cases are pending.

Fees for Medical Examinations.

Medical Examination of Candidates for Entrance to the Public Service.—The fee has been increased from 10s. 6d. to £1 1s.

Life Insurance Examination Fees.—Negotiations are still proceeding with the Insurance Offices of Australia, and up to date the only achievement on behalf of the profession has been the introduction of a simplified insurance form in which the personal questions are filled in by the proponent.

Medical Witness Fees: Post-Mortem Examination.—Under date April 5, 1948, a schedule of increased fees to be paid to medical witnesses was gazetted. The regulations under the *Coroner's Act* have also been amended, whereby the fees payable for post-mortem examination, at the request of the coroner, are £1 1s. for a superficial examination and £3 3s. for examination and making a report. These increases were the result of an approach made by the Council.

Salaries of Nurses Employed in Doctors' Surgeries.

Salaries of trained nurses which are governed by the Nurses' Award, are subject to increases in the basic wage.

Teachers' Medical Entrance Certificates.

As the form in use was unsatisfactory the matter was taken up with the authorities concerned. Advice has been received from the Department of Public Instruction that these forms have been discontinued and replaced by the health certificate used for appointments to the State Public Service.

General Practitioner Group.

The General Practitioner Group of the Branch has been resuscitated and is taking an active interest in all matters which concern general practitioners. The constitution was altered to increase the number of the committee to eight. The personnel of the committee is as follows: Chairman, Dr. L. P. Winterbotham; Honorary Secretary, Dr. Garth May; Committee, Dr. B. N. Adsett, Dr. R. S. Cohen, Dr. A. D. A. Mayes, Dr. C. T. Underwood, Dr. J. G. Wagner, Dr. E. Lorimer Walker.

Sections for the Study of Special Branches of Medical Knowledge.

Anæsthesia.—Dr. Arnold Robertson, Queensland State representative.

Ophthalmology.—Dr. F. Garret Scoles, President; Dr. J. L. Dart, Honorary Secretary.

Orthopædics.—Dr. George Douglas, Chairman; Mr. J. R. S. Lahz, Honorary Secretary.

Radiology.—Dr. Val McDowall, President; Dr. J. R. Adam, Honorary Secretary.

Workers' Compensation Insurance: Medical Fees.

The schedule of fees, as amended, came into operation on July 1, 1948. A number of anomalies, which had appeared in the original schedule, were corrected and certain doubtful points were clarified.

In the main the agreement between the Insurance Commissioner and the Council is working satisfactorily to both parties. Six cases have been submitted to the Medical Fees Tribunal for adjudication.

Members and the Insurance Commissioner owe a debt of gratitude to Dr. H. W. Horn, who acts as liaison officer, and also to the members of the Medical Fees Tribunal for their services in assisting in straightening out difficulties and dealing with complaints.

Re Railway Employees.—This matter was taken up by the Council and information was received from the Railway Department to the effect that approval has been given to revert to the previous practice of the State Government Insurance Department paying medical fees on the occasion of the first visit, and medical practitioners have been requested to issue Form 5 in all cases claiming fees from the Insurance Commissioner regarding injured railway employees.

Hospitals.

Part-Time Hospital Appointments in Country Districts.—Consideration is being given to the question of the conditions of part-time appointments at country hospitals, particularly those where medical officers have to make periodic visits to out-patient clinics in nearby towns. A circular embodying a *questionnaire* was sent to all local associations and to hospital medical officers holding part-time appointments. The replies received have been analysed by a subcommittee and steps will be taken as soon as possible to discuss the matter with the government bodies concerned, in an endeavour to have conditions improved and made uniform.

The Director-General of Health and Medical Services and the Under Secretary, Department of Health and Home Affairs, have been most cooperative and helpful.

Repatriation Commission.

Medical Benefits: Widows, Orphans and Widowed Mothers of Deceased Soldiers of the 1939-1945 War.—This service, which came into operation in Queensland on January 1, 1948, appears to be working satisfactorily.

Pharmaceutical Benefits Act, 1947.

The Commonwealth Government's Act to provide "free medicine" to the people of Australia has been the means of proving the solidarity of the profession in its policy of non-cooperation, in which members were led by the Federal and Branch Councils.

On May 25, 1948, a special general meeting of the Branch was held at which 178 members were present.

The following resolution was carried unanimously: "This meeting endorses the action of the Branch and Federal Councils in opposing *The Pharmaceutical Benefits Act* in its present form and pledges itself to support the Councils in their efforts."

Prior to this, meetings of local associations were held and also meetings of metropolitan members took place in the various suburbs which were addressed by members of the Council. At all these meetings members voted unanimously in favour of supporting the Federal Council in its policy of non-cooperation. Various circulars were sent to members at the request of the Federal Council and members were kept fully informed on the subject by Branch circulars. In addition, the Branch Council arranged for publicity both by pamphlet and through the radio to keep the people advised as to the reason for the profession's attitude.

Throughout the whole campaign members of the Branch unanimously reaffirmed their adhesion to the Federal Council's policy of non-cooperation. The outcome was a very good example of the old proverb: "United we stand . . ."

National Medical Service.

An outline of the Commonwealth Government's proposals for a national medical service, which the Minister submitted to representatives of the Federal Council of the British Medical Association at a conference held in Melbourne on October 26, 1948, was given to members in a circular.

The scheme is framed to commence as a general practitioner service available to the entire population, irrespective of financial status, and will be administered by the Department of Health. The remuneration will be on a fee-for-service basis in accordance with a schedule of fees, the Government and the patient each paying 50%.

The advantages and disadvantages were pointed out to members in the circular, and they were asked to answer two questions on the subject to guide the Branch Council in its decision.

This is the most vital issue which has ever confronted the medical profession in Australia, and members should give it their full consideration and realize all the implications involved.

Affiliated Local Associations.

The annual conference of local associations and the Council was held on June 2, 1948, at which eight local associations were represented. The following represented local associations: Dr. T. R. Gregg, Cairns; Dr. P. J. Monahan, Townsville; Dr. F. C. Wooster, Rockhampton; Dr. J. A. McGree, Gladstone; Dr. J. Morris Beale, Downs and South-Western; Dr. L. McKeon, Bundaberg; Dr. C. Constable, Ayr; Dr. J. R. Adam, Mackay. Others present were Dr. W. G. Oakeley, Warwick, Dr. L. P. Winterbotham and Dr. Garth May representing the General Practitioner Group, and Dr. Ronald Winton, Assistant Editor, THE MEDICAL JOURNAL OF AUSTRALIA. The subjects discussed included *The Pharmaceutical Benefits Act*, 1947, "Part-Time Country Hospital Appointments", "Lodge Capitation Fee", "Library Facilities for Country Members", and "Mode of Election—Office Bearers and Council of the Branch".

Reports received from local associations are published hereunder, the latest to be formed being Gladstone District in May, 1948.

Ipswich and West Moreton Local Medical Association.

The local association has this year successfully continued its regular monthly meetings to hear visiting speakers. Most of our visitors have been Brisbane specialists, and local members have been very pleased to be able to meet them and to hear their comments on current medical and surgical matters.

A clinical meeting has been arranged this year, and it is hoped to make this a regular feature of our activities.

Dr. D. A. Cameron was elected President. There are fourteen town members, five country members and one associate member.

H. G. WILSON,
Honorary Secretary.

Downs and South-Western Medical Association.

Annual Report for the Year Ended September, 1948.—The annual general meeting was held on September 25, 1948, and the election of officers for the ensuing year resulted: President, Dr. M. Graham Wilson; President-Elect, Dr. A. W. L. Row; Past President, Dr. J. G. M. Beale; Honorary Secretary-Treasurer, Dr. J. W. P. Henderson; Honorary Auditors, Dr. V. R. Woodhill and Dr. C. R. Morton; Committee, Dr. G. V. Hickey, Dr. A. S. Furness, Dr. K. J. Shellshear, Dr. W. F. Machin, Dr. R. J. H. Spark, Dr. E. A. F. McDonald and Dr. A. McGregor.

Meetings: During the year eight general and five executive meetings were held. In addition, there was a combined lecture evening with the Toowoomba Branch of the Australian Dental Association.

At the general meetings lectures were given by Dr. A. E. Lee, Dr. D. Yeates, Dr. M. J. Eakin and Dr. A. G. S. Cooper, of Brisbane, to whom we are deeply grateful. The other meetings comprised a film evening, a recording of heart sounds and a successful clinical meeting amongst our own members. All have been well attended.

During the year *The Pharmaceutical Benefits Act* of 1947 was implemented. This Association pledged its support to the Queensland Branch Council and the Federal Council in their stand against the Act.

In connexion with *The Pharmaceutical Benefits Act*, Dr. Norman Sherwood and Dr. Harold Horn visited Toowoomba and addressed a meeting to which all local practitioners were invited.

Acknowledgement: Our thanks are due to the various visiting lecturers who added interest and knowledge to our meetings; also to the Toowoomba Hospitals Board and the Medical Superintendent for their courtesy in making available the board room for meetings.

We are also grateful to Dr. C. R. Morton for the use of his rooms for executive meetings, and to Dr. G. V. Hickey for acting as liaison officer in matters requiring the assistance of the Branch Council.

Membership: Our membership has remained substantially the same and stands at 25 financial members.

Official Representation: Our President represented the Downs and South-Western Medical Association at the annual dinner of the Toowoomba Branch of the Australian Dental Association last November and at the conference of the local associations and Queensland Branch Council held in Brisbane on June 2, 1948.

J. G. MORRIS BEALE, President.
J. W. P. HENDERSON, Secretary.

Gladstone and District Local Medical Association.

Annual Report.—I have to submit the following report of the activities of this local association.

The Gladstone and District Local Medical Association came into being on May 28, 1948, when the Council of the Queensland Branch of the British Medical Association approved its formation. The following are members: Walter Yum, Roseberry Street, Gladstone (President); John Allen McGree, Bramston Street, Gladstone (Honorary Secretary); Godfrey James Byrne, Roseberry Street, Gladstone; Clive W. Kingston, General Hospital, Gladstone; James Oxley, Biloela.

To celebrate the occasion of the formation of the Association, the members and their wives entertained Dr. R. Palmerston Rundle, representing the Rockhampton Local Medical Association, and Mrs. Rundle at dinner on the night of June 11.

Work in connexion with the local association has been mostly of an organizational nature, although it was possible to have a representative present at the conference of local associations in June. It is intended to hold clinical meetings shortly, and to put to good use the helpful offers of the Post-Graduate Medical Education Committee regarding visiting lecturers.

JOHN A. MCGREE,
Honorary Secretary.

Nambour Local Medical Association.

At the annual general meeting of the Namour District Local Association held in September the new office bearers were elected as follows: President, Dr. G. E. W. Streeten, of Palmwoods; Secretary-Treasurer, Dr. A. W. Eklund, of Nambour. The membership of the Association totalled thirteen, diminished in November by the departure of one of us to twelve.

Regular business meetings were held throughout 1947 and matters dealt with were in the main *The Pharmaceutical Benefits Act*, on which subject we were addressed on two occasions by Dr. Alan Lee, and matters appertaining to local hospitalization difficulties. The latter difficulty has been solved by the opening of a very satisfactory private hospital in Nambour. With regard to *The Pharmaceutical Benefits Act* all practitioners in this area are unanimous in their support of the Branch Council attitude.

Regular clinical meetings and film evenings have been held throughout the year. We are indebted to Wyeths Incorporated and Taylors Elliotts Proprietary, Limited, for the films shown and also to Mr. E. McCorkell, proprietor of the Vogue Theatre, Nambour, who showed the films in his own theatre under the best possible screening conditions.

Lectures, arranged through the Post-Graduate Medical Education Committee, have been given by numerous lecturers including Dr. A. E. Lee, Dr. A. V. Meehan, Dr. D. A. A. Davis, Dr. B. B. Barrack, Dr. Phil Macindoe and Dr. H. S. McLelland. Two clinical meetings have been held by practitioners, relying upon local material for the subject matter.

Altogether the year has been a successful one, and despite many difficulties, such as poor attendance due to inability through exigencies of practice, weather and the like, the local association continues to flourish and has, if anything, increased in strength.

A. W. EKLUND.
Honorary Secretary.

Townsville Local Medical Association.

I have to report that activities of the Townsville Local Association have been characterized by enthusiastic interest of the members in both clinical and medico-political affairs.

Meetings have been held fortnightly throughout the year, and programmes were arranged consisting of the reading of papers, clinical demonstrations and the discussion of medico-political and general business matters.

The annual general meeting of the Branch was held on January 22, 1948. Dr. W. B. Chapman was reelected President for the year 1948, and Dr. J. Breinl, Honorary Secretary-Treasurer. A committee consisting of Dr. T. U. Ley, Dr. P. Monahan, Dr. G. Moore and the Honorary Secretary was formed to arrange a programme of activities for the year. The Branch was privileged to enjoy visiting lecturers sponsored by the Post-Graduate Medical Education Committee. The visiting lecturers on each occasion spent two days in Townsville. Clinical demonstrations and lectures were followed by most successful dinners held in the Queens Hotel. Dr. K. Hirschfeld (surgery) and Dr. D. C. Jackson (pædiatrics) visited Townsville in June, and Dr. H. Love (medicine) and Dr. A. J. Foote (dermatology) visited this city in October. These visits by post-graduate

teachers were much appreciated, but they are all too short and too infrequent.

Unfortunately few medical men from surrounding towns seem able to spare time to attend these lectures. This appears unavoidable on account of long distances and a large number of "one man" towns.

Dr. A. Shaw, of the Queensland Red Cross Blood Transfusion Service, addressed this Branch in May, the subject being "Blood Transfusion" and "Intravenous Therapy Techniques". In September, Mr. R. Corbett, of London, read a paper to this Branch on "Arrested and Ectopic Testes", and later in that month, Dr. F. Tod Stevens, of Melbourne, read a paper on "Recent Advances in Medicine".

Clinical meetings were held as follows:

February 5, 1948: Paper presented by Dr. King on "Prophylaxis of Communicable Diseases in Childhood". Clinical cases by Dr. King and Dr. A. Fraser.

February 19, 1948: Paper presented by Dr. T. U. Ley on "Tumours of Bone".

March 4, 1948: Clinical cases—Dr. King, Dr. Moore and Dr. Remington.

April 15, 1948: Clinical cases—Dr. V. F. O'Neill and Dr. J. Breinl.

May 27, 1948.—Clinical cases—Dr. A. Fraser.

June 10, 1948: Showing of medical films by courtesy of Wyeths Incorporated—(i) "Anatomy of the Abdominal Wall", (ii) "Lower Segment Cæsarean Section".

August 5, 1948: Paper presented by Dr. T. U. Ley on "Peripheral Nerve Lesion of the Upper Arm".

October 28, 1948: Showing of films from the library of the Post-Graduate Medical Education Committee of New South Wales—(i) "Intravenous Anæsthesia", (ii) "Cholecystectomy".

Dr. P. J. Monahan was elected to represent the Townsville Local Association at the conference in Brisbane in June of this year.

I am pleased to report that this Branch has unanimously supported the policy of the British Medical Association in regard to *The Pharmaceutical Benefits Act*. With very rare exceptions the public of North Queensland have not questioned in any way the stand adopted by their medical advisers.

Members of this Association have been active in regard to the Townsville Branch of the Bush Children's Health Scheme. They have given ungrudging service in the cause of approximately 150 outback children who have passed through the Bush Children's Home during the year.

The annual subscription to the local association was fixed at £2 2s. for ordinary members and £1 1s. for junior resident medical officers.

Membership of the Branch now numbers twenty-one.

I wish to thank all members of the Branch for their support and enthusiasm, which have been entirely responsible for the success of the year's activities.

W. G. CHAPMAN,
President.

Mackay Local Medical Association.

At the annual general meeting held on September 7, 1948, Dr. C. E. Williams was reelected President and Dr. S. C. Williams as Secretary-Treasurer of the Mackay Local Association. The annual report and balance sheet were read and approved.

Quarterly meetings of the Association were held during 1947-1948, each with an almost 100% attendance. A special meeting was held in October, 1947, to discuss and support Federal Council dealings with the Minister of Health.

Routine matters were discussed at quarterly meetings and special discussions were held on legacy, lodge practice, part-time paid medical officers and *The Pharmaceutical Benefits Act*.

Visiting lecturers during the year were Dr. K. Hirschfeld, Dr. D. Jackson and Dr. A. E. Shaw.

Wyeth's representative showed a film on "Low Cæsarean Section".

Local lecturers at quarterly meetings were: Dr. Ian Chenoweth, "Physiological Aspects of the Female Sex Cycle and Practical Applications"; Dr. Paul Hopkins, "Rheumatoid Arthritis and Infective Arthritis"; Dr. Robert Grant, film and talk on "Breech Presentations"; Dr. George Bolton, "Asphyxia Neonatorum".

The Mackay Local Medical Association has fourteen members.

S. C. WILLIAMS,
Honorary Secretary.

Rockhampton Local Association.

During the year interest has been well maintained. Monthly clinical meetings have been well attended, and in this regard we owe much to the medical superintendent of our General Hospital for his keenness and work in arranging these meetings. We have appreciated sincerely the visits of those medical men sent by the Post-Graduate Committee and have gained much benefit from their lectures and demonstrations.

The Pharmaceutical Benefits Act has been given considerable attention and this Association will adhere strictly to the letter of our decision "not to cooperate"—at the same time we will appreciate always the advice of our State Council in matters of such importance. A canvass of the local chemists discloses that no government form for free medicine has been received by them.

Our membership, including medical men from neighbouring towns, stands at thirty.

E. R. WATKINS,
Honorary Secretary.

Cairns Local Association.

The Cairns Local Medical Association has been more active during this last twelve months. Regular clinical meetings have been held each month and interest has been further stimulated by visits from the representatives of the Post-Graduate Medical Education Committee.

At a special meeting it was resolved to uphold the decisions of the Federal Council of the British Medical Association in its discussions on "free medicine" legislation.

C. H. KNOTT,
Honorary Secretary.

No annual reports have been received from the following local associations: Bundaberg, Maryborough, Lower Burdekin (Ayr), South Burnett.

Royal Australasian College of Physicians.

The annual meeting of the Royal Australasian College of Physicians was held this year on June 10, 11 and 12. This was the first occasion on which the annual meeting was held in Brisbane. Fellows and members from other States also attended.

There was a dinner for Fellows and Members at Lennon's Hotel at midday on June 10 and in the afternoon the first scientific session took place. A late afternoon party was held at Lennon's Hotel at 5.45 p.m. to which Fellows, Members and their wives were invited.

On the afternoon on June 11 the second scientific meeting was held. In the evening, in the hall of the Brisbane Boys' Grammar School on Gregory Terrace, in the presence of Fellows and Members, their wives and many invited guests, Professor Wadham, of Melbourne University, delivered the Annie B. Cunning Oration, the subject being "The Humus Controversy". At the conclusion of the oration supper was served.

The last event was the holding of a clinical meeting on the morning of June 12.

Royal Australasian College of Surgeons.

An invitation was extended to members of the Branch by the Queensland State Committee of the Royal Australasian College of Surgeons to attend an address given by the Commonwealth Travelling Professor, Sir Hugh Cairns, K.B.E., D.M. (Oxon.), F.R.C.S., on "The Treatment of Pyogenic Meningitis" on March 19.

University of Queensland.

British Medical Association (Queensland Branch): Queensland Medical Students' Loan Fund.—The personnel of the committee of administration is as follows: Dr. C. A. Thelander (chairman), the Dean of the Faculty of Medicine (Dr. E. S. Meyers), Professor Alex. Murphy, Dr. Arnold Robertson and a representative of the University of Queensland Medical Society (nominated annually by the society), and the ex-officio members of the Council. The fund now stands at £585 10s. 4d. During the year £61 5s. 3d. was donated by members of the Branch.

Honorary Associate Members of the Branch.—It is pleasing to note that there are now 128 fourth, fifth and sixth year medical students enrolled as honorary associate members of the Branch, 21 of whom were elected during the year.

University of Queensland Medical Society.—Dr. W. H. Steel acts as liaison officer between the Council and the society. During the year the society again approached the Council regarding the inauguration of a student vacation apprenticeship scheme and asked the Council to approve of the principle. A reply was sent stating that the Council had previously approved of the principle and members of the

Association have been informed. The approach to individual doctors must come direct from the society's members.

Post-Graduate Medical Education Committee.—The representatives of the Branch on this committee are Dr. Felix Arden, Dr. Harold R. Love and Dr. Alan E. Lee. The Post-Graduate Committee is very anxious to help in any way it can by sending lecturers to centres. During the year thirteen centres have been visited by twenty-five lecturers; Professor J. C. Spence, Nuffield Professor of Child Health, University of Durham, and Mr. Rupert Corbett, Surgeon to Saint Bartholomew's Hospital, London, were the 1948 visitors from overseas. Shortly medical films will be available to country centres through the Queensland Committee by courtesy of the New South Wales Committee which has built up a collection of some 160 films.

Post-Graduate Week was held from May 28 to June 4, 1948. The visiting interstate lecturers were Dr. Mary Heseltine, Professor Bruce Mayes and Dr. Douglas Miller.

The Memorial Prize of the British Medical Association (Queensland Branch), Harold Plant Memorial Prize and William Nathaniel Robertson Medal.—Advice has not yet been received from the Registrar of the University of Queensland regarding these prizes which have not yet been awarded.

Professor E. J. Goddard Memorial.—The President, Dr. Norman Sherwood, represented the Branch on the committee of the E. J. Goddard Memorial Fund. Members of the Branch were asked to subscribe to the fund in recognition of the valuable assistance given by the late Professor Goddard in the establishment of the Faculty of Medicine within the University of Queensland.

Federal Council of the British Medical Association in Australia.

Meetings of the Federal Council were held in Melbourne from March 11 to 13, and on August 12 to 15 and 20, 1948, in Perth. In addition special meetings were held to deal with *The Pharmaceutical Benefits Act* in Sydney on May 15 and 16, and in Melbourne on July 3. At all these meetings the Branch was represented by Dr. Alan E. Lee and Dr. H. W. Horn.

A conference of representatives of the Federal Council took place on October 26, 1948, in connexion with the Commonwealth Government's plan for a national medical service.

Full-Time Secretariat.—The Federal Council has decided to appoint a full-time assistant secretary to the General Secretary.

Federal Medical War Relief Fund.

The Local Committee of Management will be glad to hear of dependants of deceased or incapacitated medical officers of the 1939 war who are eligible for assistance from the fund. Steps have been taken to ascertain this information from the headquarters of the services.

Code of Medical Ethics.

Code of Medical Ethics, Federal Council of the British Medical Association in Australia.—A code of Medical Ethics has been published by the Federal Council as principles and rules suggested as general guides. It is left to the Council of each Branch to decide individual cases on their merits and special circumstances and to determine the penalty of any infringement of ethical principles.

"The Medical Journal of Australia."

In June last the Branch was pleased to welcome the Assistant Editor, Dr. Ronald Winton, who paid his first visit to Brisbane in his official capacity and renewed many friendships.

Australasian Medical Publishing Company, Limited.

The company has suffered a great loss in the death of Dr. T. W. Lipscomb, who was Chairman of Directors. A letter of condolence was sent to the directors. A further great loss was caused by the death of Dr. D. Gifford Croll, a director and representative of the Queensland Branch on the Board of Directors for many years. Dr. Alan Lee, previously a member of the committee, was appointed to the Board of Directors to fill the vacancy and Dr. H. W. Horn was appointed a member of the company. Dr. T. A. Price is also a member of the company.

Australasian Medical Congress, Sixth Session, Perth, 1948.

The first session of the Australasian Medical Congress since the war was held in Perth from August 15 to 21, 1948, and was a most successful event both scientifically and socially.

Dr. Harold Horn was appointed Vice-President of Congress, representing the Queensland Branch, and Dr. Harold Love acted as Honorary Local Secretary.

Australasian Medical Congress, Seventh Session, Brisbane, 1950.

The Federal Council has accepted the invitation of the Queensland Branch to hold the seventh session of the Australasian Medical Congress in Brisbane in 1950, and steps have been taken to initiate the organization by the formation of the executive committee and the appointment of officers.

The full support of every member of the Branch will be necessary to make the congress a success.

British Medical Association Advisory Bureau.

The Council of the British Medical Association has established the Medical Advisory Bureau in London, with a view to welcoming and providing an advisory service to medical practitioners visiting the United Kingdom, particularly those from dominions and colonies. This should prove of great value to members of the Association.

British Commonwealth Medical Council.

The Queensland Branch Council expressed itself as being highly in accord with the desirability of accepting the invitation of the British Medical Association for Australia to be represented at a meeting to consider the formation of a British Commonwealth Medical Association to be held in London on September 15 and 16, 1948.

The British Medical Association in Australia was represented by Dr. A. J. Collins, Dr. Mervyn Archdall and Dr. Athol Quayle, a member of the Queensland Branch.

At this meeting it was suggested that the conference in 1950 be held in Brisbane in conjunction with the Australasian Medical Congress, because the British Commonwealth Medical Council desires to meet in conjunction with conventions in host countries.

World Medical Association.

This organization has been functioning for two years and *questionnaires* have been answered by the Branch Council on many matters dealing with the practice of medicine generally.

Dr. A. J. Collins and Dr. Mervyn Archdall represented the Federal Council of the British Medical Association in Australia at the second annual meeting which was held in Geneva from September 8 to 11, 1948.

These activities indicate a widening of the organization of the profession, which has always been scientifically universal.

Indemnity Insurance.

By amendment of its Articles of Association the Medical Defence Society of Queensland has made it obligatory as from January 1, 1949, for its members to indemnify themselves through the Medical Protection Society, Limited (or such other person or corporation as may from time to time be approved by its Council), with regard to actions, proceedings, claims and demands against them as affecting their professional character or interests, for a sum not less than one thousand pounds sterling in any one case and two thousand pounds sterling in any one year.

British Medical Agency of Queensland, Proprietary, Limited.

The Agency continues in its valued service to members and to the Association. The relative shortage of doctors in Queensland available for locum duties, assistantships and partnerships throughout the year, and particularly the latter portion, has militated against members' receiving the full assistance formerly obtained.

The financial results are reported to be satisfactory. The Association is appreciative of the work done in its interests by the manager and his staff.

Queensland Medical Finance Proprietary, Limited.

The Queensland Medical Finance Proprietary, Limited, operated on a larger scale during the past year than at any time since 1940.

It is expected that, as general conditions become more normal, a greater number of members will seek financial assistance from the company.

Social.

The British Medical Association annual ball took place on Wednesday, June 2, in Lennon's ballroom. Dr. Robert S.

Morison, Assistant Director of the Medical Sciences Staff of the Rockefeller Foundation, and Dr. Ronald Winton, Assistant Editor of THE MEDICAL JOURNAL OF AUSTRALIA, were guests. There was a good attendance of members and a very enjoyable evening was spent.

The committee responsible for the arrangements were Dr. Horace Johnson, Dr. Harold R. Love and Dr. Arnold W. Robertson.

The Council dinner this year was held on Wednesday, November 10, at the National Hotel. Guests included the Bancroft Orator (1948), Dr. E. H. Derrick, Dr. P. A. Earnshaw, representing the Royal Australasian College of Physicians, and Dr. Alan E. Lee, representing the Royal Australasian College of Surgeons.

Conclusion.

This year has been crammed full of activity. It witnessed the attempt to implement *The Pharmaceutical Benefits Act* in a form which was unacceptable to the profession. The complete unity of the members of the British Medical Association has given a clear demonstration of what can be achieved when we speak with one voice. Even more vital decisions will have to be made in the near future. If the profession maintains its unity there is nothing to fear in any future negotiations. If we wish to maintain our much cherished freedom to practise medicine without governmental control we must all unanimously support the decisions made by our elected representatives.

A great debt of gratitude is due to our Federal Council representatives, Dr. Harold Horn and Dr. A. E. Lee, for their untiring services to the Branch during the past year.

Mrs. Spooner and the office staff have had a very busy year. Our unstinted thanks are given to them for the highly efficient manner in which the affairs of the Branch are managed.

I would like to express the appreciation of the Branch to the Director-General of Medical Services and to the Under Secretary of the department for their helpful cooperation during the year.

(Sgd.) NORMAN SHERWOOD,
President.

QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION.
(INCORPORATED.)

Balance Sheet as at November 15, 1948.

LIABILITIES.	£ s. d.	£ s. d.
Fixed—		
Loan from Queensland Medical Land Investment Company, Limited		4,650 0 0
Current—		
Subscriptions for Remittance to—		
British Medical Association, London	29 19 3	
Australasian Medical Publishing Company, Limited, Sydney	40 2 6	
		70 1 9
Association Funds—		
Sinking Fund	430 13 8	
Superannuation Fund	467 6 8	
	898 0 4	
Accumulation Fund	7,563 12 6	
Reserve for Entertainments	25 4 2	
		8,486 17 0
		£13,206 18 9

ASSETS.	£ s. d.	£ s. d.
Fixed, at cost, *less* depreciation—		
Land and Buildings—B.M.A. House	2,296 2 0	
Bartley Street Property—at cost	1,733 11 5	
Architect's Fees—preparing plans for prospective new building—at cost	300 0 0	
Library	150 0 0	
Typewriters, Book-cases, Balopticon and Furniture	61 1 5	
Bancroft Medals and Collar	4 10 0	
		4,545 4 10
Queensland Medical Land Investment Company, Limited—5,950 shares of £1 each paid to 10s. each at cost	2,975 0 0	
British Medical Agency of Queensland Proprietary, Limited—258 shares of £1 each, fully paid, at cost	258 0 0	
		3,233 0 0
Australasian Medical Publishing Company, Limited, Sydney—		
5% Debentures—at cost	55 0 0	
3¾% Series "E" Debentures—at cost	500 0 0	
Advance not yet converted to Series "E" Debentures	88 18 3	
		643 18 3
		8,422 3 1
Current—		
Australian Consolidated Inscribed Stock at cost—		
3¼%, maturing 1959 £1,500 0 0		
3¾%, maturing 1960 £300 0 0		
	1,800 0 0	
English, Scottish and Australian Bank, Limited	1,767 7 7	
Sundry Debtors	312 0 0	
Electric Light Deposit	6 0 0	
Cash	1 7 9	
		3,886 15 4
Fund Investments—		
Sinking Fund—		
Australian Consolidated Inscribed Stock—		
£280, 3¾%, maturing 1951—at cost	278 3 3	
£90, 3¼%, maturing 1960	90 0 0	
Commonwealth Savings Bank, Brisbane	62 10 5	
	430 13 8	
Superannuation Fund—		
Commonwealth Savings Bank, Brisbane	467 6 8	
		898 0 4
		£13,206 18 9

We have compared the above Balance Sheet with the books, accounts and vouchers of the Queensland Branch of the British Medical Association (Incorporated), and have obtained all the information and explanations we have required. The title deeds of the Bartley Street Property are not yet available for our inspection.

Subject to this, in our opinion, the Balance Sheet is properly drawn up to exhibit a true and correct view of the state of the Association's affairs as at November 15, 1948, according to the best of our information and the explanations given to us, and as shown by the books of the Association.

The Register of Members and other records which the Company is required to keep by the Companies Acts of 1941-1942, or by its Articles, have, in our opinion, been properly kept.

R. G. GROOM & COMPANY,
Chartered Accountants (Aust.),
Auditors.

Brisbane, November 26, 1948.

(Sgd.) H. W. HORN,
Honorary Treasurer.

QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION.
(INCORPORATED.)

Revenue Account for Twelve Months ended November 15, 1948.

EXPENDITURE. November 15, 1948.	£ s. d.	£ s. d.	REVENUE. November 15, 1948.	£ s. d.	£ s. d.
To Branch Expenses	1,493 19 6		By Branch and Organization Fund Subscriptions		2,889 19 5
„ Library Expenditure	142 18 4		„ Portion of Journal Subscriptions to be applied to Australasian Medical Publishing Company, Limited, Sydney, Series "E" Debentures		588 18 3
„ Depreciation of Office Equipment	7 6 10				3,478 17 8
		1,644 4 8	„ Interest—		
„ Provision for Staff Superannuation		60 0 0	Commonwealth Government Inscribed Stock	58 10 0	
„ Federal Council— Contribution to Organization Expenses		537 15 0	Australasian Medical Publishing Company, Limited, Debentures	17 4 5	
„ Net Surplus for Year— Transferred to Accumulation Account		1,369 12 6			75 14 5
			„ General— Sale of Nutrition Pamphlets and Lists of Members		8 6 10
			„ Rents	356 17 0	
			Less Expenses	308 3 9	
					48 13 3
		£3,611 12 2			£3,611 12 2

QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION.
(INCORPORATED.)

Statement of Receipts and Payments for Twelve Months ended November 15, 1948.

RECEIPTS. November 16, 1947.	£ s. d.	£ s. d.	PAYMENTS. November 15, 1948.	£ s. d.	£ s. d.
To Funds at November 16, 1947—			By Amounts remitted on account of Subscriptions collected to—		
English, Scottish and Australian Bank, Limited — Current Account	2,091 15 1		British Medical Association, London	930 2 0	
Cash in Hand	4 12 10		Australasian Medical Publishing Company, Limited	793 7 6	
		2,096 7 11			1,723 9 6
November 15, 1948.			„ Federal Council— Contribution to Expenses		537 15 0
To Subscriptions—			„ Branch Expenses—		
Queensland Branch Subscriptions	1,574 10 11		Salaries, Audit and Honoraria	804 18 4	
Organization Fund, Queensland Branch	1,258 8 6		Publicity *re Pharmaceutical Benefits Act*	335 7 7	
For remittance to British Medical Association, London	933 18 9		Printing and Stationery	137 10 6	
For remittance to THE MEDICAL JOURNAL OF AUSTRALIA, Sydney	791 10 0		Postages and Duty Stamps	115 9 7	
		4,558 8 2	Bank Charges, Meeting Expenses and Sundries	57 7 4	
„ General—			Telephone	25 12 9	
Rents	106 17 0		Gas and Lighting	17 13 5	
Interest on Commonwealth Inscribed Stock	58 10 0				1,493 19 6
Australasian Medical Publishing Company, Limited—Interest on Debentures	17 4 5		„ B.M.A. House Expenses—		
Sale of Nutrition Pamphlets and Sundries	8 6 10		Rates to December 31, 1948	145 6 0	
Sale of Gestetner Duplicating Machine	5 0 0		Cleaning	93 17 5	
		195 18 3	State Land Tax	10 18 9	
			Repairs and Maintenance	10 2 8	
			Insurance	6 0 9	
					266 5 7
			„ Bartley Street Property— Balance of Purchase Price		850 0 0
			„ General—		
			Library Expenditure	142 18 4	
			Transfer to Superannuation Fund Account with Commonwealth Savings Bank	60 0 0	
			Net Cost—British Medical Association Ball	7 11 1	
					210 9 5
			„ Funds at November 15, 1948—		
			English, Scottish and Australian Bank, Limited, Brisbane	1,767 7 7	
			Cash	1 7 9	
					1,768 15 4
		£6,850 14 4			£6,850 14 4

BALANCE SHEET AND FINANCIAL STATEMENT.

The balance sheet and financial statement as at November 15, 1948, were received and adopted on the motion of Dr. Harold Horn, the Honorary Treasurer, seconded by Dr. Ellis Murphy. The financial statement is published herewith.

AMENDMENT OF BY-LAW.

A notice for the amendment of By-Law 4, due notice of which had been given, was proposed by Dr. Harold Horn, seconded by Dr. W. J. Saxton and carried unanimously. It was explained that increases in the subscription rate had become necessary, largely as a result of higher Federal Council expenses. Under the amended by-law the subscriptions for 1949 would be as follows: senior metropolitan members, £9 9s.; senior country members, £8 8s.; metropolitan professional officers, £7 7s.; country professional officers, £6 6s.; university staff, £6 6s.; junior members (under three years' qualification), £4 4s.; retired members, £4 4s.

MEMORIAL ROLL.

With the members standing, the President read the names on the Memorial Roll of the Branch.

ETHICS COMMITTEE.

Dr. V. McDowall, Dr. J. J. Power, Dr. M. Graham Sutton, Dr. J. G. Avery, Dr. L. J. J. Nye and Dr. J. G. Wagner were

reelected members of the Ethics Committee. Dr. F. W. R. Lukin was elected to fill the vacancy caused by the death of Dr. D. Gifford Croll.

ELECTION OF OFFICE-BEARERS.

The President announced the results of the election of office-bearers and members of the Council.

President: Dr. B. L. W. Clarke.
President-Elect: Dr. Harold R. Love.
Past President: Dr. Norman Sherwood.
Honorary Secretary: Dr. J. R. Adam.
Councillors: Dr. Felix Arden, Dr. J. W. Best, Dr. A. W. Eklund, Dr. K. B. Fraser, Dr. Glen V. Hickey, Dr. H. W. Horn, Dr. A. E. Lee, Dr. A. D. A. Mayes, Dr. H. S. Patterson, Dr. Athol Quayle, Dr. R. G. Quinn, Dr. Arnold Robertson, Dr. E. Lorimer Walker.

ELECTION OF AUDITORS.

Messrs. R. G. Groom and Company were reelected auditors for the ensuing year.

INDUCTION OF PRESIDENT AND PRESIDENT'S ADDRESS.

Dr. Norman Sherwood vacated the chair in favour of Dr. B. L. W. Clarke, the incoming President. Dr. Clarke paid a tribute to the work of Dr. Sherwood and delivered his president's address (see page 253).

VOTES OF THANKS.

Votes of thanks were passed to the retiring Honorary Secretary (Dr. Arnold Robertson), the out-going President (Dr. Norman Sherwood), the Council for 1948 and to the Secretary, Mrs. Spooner, and her staff.

Medical Societies.

THE PUBLIC MEDICAL OFFICERS' ASSOCIATION OF NEW SOUTH WALES.

THE annual general meeting of the Public Medical Officers' Association of New South Wales was held at British Medical Association House, 135 Macquarie Street, Sydney, on Thursday, January 27, 1949. Fifteen members were present.

Annual Report.

The following annual report was adopted.

Unsatisfactory economic and industrial conditions generally have continued the difficulties under which the majority of public medical officers endeavour to carry out their duties. Unfortunately there are no signs that any amelioration of conditions is likely in the near future.

The membership roll of the Association now contains 133 members, including nine honorary members.

It is with great regret that we record the death of Dr. D. Wallace and Dr. S. Evan Jones. The latter was for many years a member of the committee and one whose opinions were greatly valued. A donation of £50 has been made from the funds of the Association to the S. Evan Jones Memorial Fund.

During 1948 the committee met eight times, attendance of members being as follows: Dr. E. T. Hilliard (chairman), 8; Dr. G. Cuthbert, 4;[1] Dr. G. B. Wooster, 6; Dr. E. Wallace, 7; Dr. J. McManamy, 6; Dr. C. England, 7; Dr. D. McClemens, 5; Dr. W. Flook, 7; Dr. G. Saxby, 4; Dr. A. T. Edwards, 7; Dr. Sandes, 5; Dr. N. Kirkwood, 5; Dr. Main, 2; Dr. V. Brown, 3; Dr. S. Repin, 3.[1]

The chief efforts of the committee during the year were devoted to an endeavour to obtain a satisfactory salaries agreement with the Public Service Board. These efforts met with partial success, the resulting salaries representing a definite increase on previous rates, but failing to provide the salaries to which the committee considered members were entitled. Especially is it deplored that the rates of pay for female medical officers remain at such a low level.

E. T. HILLIARD, President.
A. T. EDWARDS, Honorary Secretary.

Election of Officers.

The election of officers resulted as follows:

President: Dr. J. Flook.
Honorary Secretary: Dr. N. Kirkwood.
Honorary Auditor: Dr. J. McGeorge.

Committee: Dr. G. Cuthbert, Dr. J. McManamey, Dr. G. Smith, Dr. J. Landor, Dr. S. Repin, Dr. D. McClemens, Dr. E. T. Hilliard, Dr. G. B. Wooster, Dr. G. Ross, Dr. G. Sandes, Dr. G. C. Saxby.

[1] Elected March, 1948.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR MARCH.

Classes for Higher Degrees and Diplomas.

THE following classes at the University of Melbourne for higher degrees and diplomas (Part I M.D., M.S., D.O., D.L.O., D.G.O., D.D.R., D.T.R.E., D.P.M. and D.A., and pathology for Part II M.S. and diplomas) will commence on Wednesday, March 16, 1949, and continue till July or August.

Monday and Wednesday afternoons: 1 to 2.30 p.m., pathology practical demonstration; 2.35 to 3.40 p.m., pathology lectures; 2 to 3.15 p.m, anatomy lectures; 3.45 to 5 p.m., physiology lectures.

Thursdays: 3.45 to 5 p.m.: physics lectures.

Classes will be held only if there are sufficient enrolments for the degree or diploma concerned to warrant a course.

The fees will be £31 10s. as the total amount for courses for Part I of a degree or diploma, £15 15s. for each subject taken separately.

Lectures in gastro-intestinal disorders suitable for candidates for the M.D. Part II and M.R.A.C.P. will be given by Dr. M. V. Clarke at 2 p.m. on the following dates at Saint Vincent's Hospital: February 28, "Diarrhœa"; March 7, "Gastro-Intestinal Hæmorrhage". The fee will be £1 1s. Dr. D. C. de Gruchy will assist with clinical and pathological demonstrations.

Clinical lectures and demonstrations in dermatology arranged by the British Association of Dermatology and Syphilology will be conducted on Tuesday and Thursday afternoons, commencing at 2 p.m. on March 1, 1949. The fee will be £5 5s.

Enrolments for the above three courses should be made with the Secretary of the Committee, 426 Albert Street, East Melbourne, JM 1547.

Clinic at Eye and Ear Hospital.

A post-graduate clinic will be held at Dr. Walter Williams's ear, nose and throat clinic at the Eye and Ear Hospital on Monday, March 7, 1949, at 4.30 p.m.

Country Courses.

The following country courses have been arranged.

Geelong: Saturday, March 5, 1949. Enrolments should be made with Dr. M. W. Morris, "Belleville", Ryrie Street, Geelong. Telephone 4567.

Mooroopna: Saturday and Sunday, March 19-20, 1949. Enrolments should be made with Dr. B. R. Schloeffel, Maud Street, Shepparton. Telephone Shepparton 67.

Flinders Naval Depot: Wednesday, March 9, 1949, at 2.30 p.m. Dr. Eric Price will give a demonstration on "Osteomyelitis and Bone Diseases" by arrangement with the Royal Australian Navy.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Armidale.

THE Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Northern District Medical Association, will hold a week-end course at the New England University College, Armidale, on Saturday afternoon and Sunday, March 5 and 6, 1949. Dr. Lorimer Dods, Dr. F. A. Maguire and Dr. I. Douglas Miller will take part. The fee for the course will be £2 2s. Those wishing to attend are requested to communicate with Dr R. J. Jackson, Honorary Secretary, Northern District Medical Association, Armidale, as soon as possible.

Correspondence.

A NATIONAL HEALTH SERVICE.

SIR: Your recent publication of *The National Health Service Act* and of the parliamentary discussions relating thereto is timely and has given the medical profession an opportunity to survey the whole proceedings.

My own opinion is that the medical profession should not accept this *National Health Service Act* as constitutional; in many respects it could quite probably be successfully held to be illegal.

The social services questio of the referendum of 1946 asked the people of Australia to vote "yes" or "no" as to whether they desired the Comnonwealth Parliament to make laws "with respect to the provision of medical and dental services"; the people voted in the affirmative. It is to be noted that the word "provision"only was used. If the people had been asked to vote "with respect to the provision and control of medical and dental services", the voting could have been in the negative.

The people of Australia thn have given the Federal Government permission to "rovide" a medical service financially; the Government ha not been given any power whatever to control, conduct, administer or manage a medical service, medical equipment or hospitals, laboratories, health centres or clinics.

Once the Federal Governmet provides all these things, someone, of course, has to conrol and conduct them, such as a corporate body or commission, but not the Federal Government itself.

Surely the English language annot be so distorted as to successfully contend that the vord "provision" means or includes "control".

Yours, etc.,
A. LESLIE WATSON.

Sydney,
January 7, 1949.

OBSERVATIONS ON ONE HUNDRED AND SIX CASES OF RECURRENT INUINAL HERNIA.

SIR: Dr. Wyndham is to be cgratulated on his article on recurrent inguinal hernia in yo issue of January 15, 1949. This article occupies only threcolumns, but the information contained in it is of the tmost importance. In his forthright manner Dr. Wyndha has stated his facts and refused to comment on them. Nertheless some comment is certainly necessary, particularl as far as they apply to recurrence after operation for flirect inguinal hernia.

It may be said that, in the tudy of recurrent inguinal hernia, there are two outstandir unsolved problems: (i) the correlation of the exact site (recurrence with the type of operation performed at the fit operation; (ii) the assessment of recurrence rates of diffent types of operation.

That these problems are unsved is due to the fact that in the past surgeons have guped together all types of recurrence under the one headi "Recurrences" without any attempt to differentiate and desibe the exact site of recurrence. It is amazing to think th the present state of affairs, whereby most complicated andideed dangerous operations are carried out as routine procures, has been reached not by a scientific study, but by iance on authority and on a vague impression. The autlity is that of Bassini and the impression is that the mainecurrence is a "direct" one.

In a recent article, to which :. Wyndham has been kind enough to refer, the writer, eaking of indirect herniæ only, has attempted to show (i that if a simple operation is performed whereby nothing se is done beyond dealing with the sac and repairing) internal ring, the main recurrence is indirect and a dct recurrence is a rarity; (b) that a direct recurrence the result of an extensive plastic operation and is therefi not a true recurrence at all; (c) that the performance an extensive plastic operation does not lessen the likelihi of an indirect recurrence.

An analysis of Dr. Wyndhas results amply bears out these suggestions, and moreove demonstrates further that even after a plastic repair tmain recurrence is not a direct one, as has always been sumed, but an indirect one. These matters will now be codered in more detail.

In 18 of Dr. Wyndham's casthere had been an excision of the sac only. In all 18 es the recurrence was an indirect one. In his own are the writer presented 21 cases of recurrence after hermmy only and of these all were indirect, with the except of one which was of the Ogilvie "tubular" or "funicularype and was probably not a recurrence at all. Taking the writer's and Dr. Wyndham's cases together, there is a series of 39 recurrences after herniotomy only and all of them with one exception are indirect. There is therefore strong evidence to support the contention that, if a simple operation is performed dealing only with the sac and the neighbourhood of the internal ring, a direct recurrence is a rarity.

If this group of 18 cases of simple herniotomy be removed from Dr. Wyndham's list there remains the other group of 88 cases. Of these the original hernia was indirect in 82 and direct in six. Dr. Wyndham was able to say that of the original operations 66 had included some method of repair of the canal, but in 22 the matter was doubtful. The recurrences analysed show that 65 were indirect and 23 direct. The presence of the six cases in which the original hernia was direct rather clouds the issue here. However, it can pretty safely be assumed (Dr. Wyndham can confirm this or otherwise) that the original operation in these cases included repair of the canal and further that the recurrence in each case was direct. If these six cases are removed from the list there is left a group of 82 cases where the original hernia was indirect, and where the type of operation included repair of the canal in 59 cases, but was doubtful in 22. The recurrences analysed show 65 indirect and 17 direct herniæ. Even allowing for the doubt about the 22 cases the incidence of 17 direct recurrences in this group forms a striking contrast to the incidence of no cases of direct recurrence in the group where simple herniotomy had been performed. Dr. Wyndham points out very rightly that in this group the surgeon may have found conditions at the original operation which suggested that a direct hernia was likely to occur and that performance of a plastic operation did not produce the direct recurrence, but merely failed to prevent it. The facts produced in regard to this matter by Dr. Wyndham must be studied in conjunction with those presented in the writer's article. It is suggested that a reasonable surgeon will come to the conclusion that there is a strong possibility that "direct" recurrences after extensive plastic operations for indirect inguinal hernia are in most instances the result of the operation.

Reference will now be made to the other striking point that is made obvious by Dr. Wyndham's analysis of the group of cases where there was a repair of the canal. Whereas there were only 17 direct recurrences in this group, there were no less than 65 indirect recurrences. This revelation should do much to dispel the vague impression that the main recurrence after plastic repairs is the direct one. Furthermore it demonstrates very vividly that the performance of a plastic repair involving the canal cannot be expected to prevent the indirect recurrence.

There is only one other point in Dr. Wyndham's article which will be referred to. His analysis of the time intervals elapsing between the date of operation and the date of recurrence makes it clear that, in order that some idea may be formed as to how a series of cases is going to turn out, an interval of at least three years must pass after the performance of the last operation in the series.

In the conclusion to his own article the writer stated that the plastic operations should be abandoned, at all events as routine procedures, and that all efforts should be transferred to the prevention of the indirect recurrence. The material presented by Dr. Wyndham has done much to strengthen this opinion.

Yours, etc.,
C. CRAIG,
Surgeon Superintendent.

General Hospital,
Launceston,
January 27, 1949.

THE OVER-EMPHASIS ON PSYCHIATRIC SYMPTOMS.

SIR: I wish to offer the congratulations of a country "G.P." to S. J. Minogue (THE MEDICAL JOURNAL OF AUSTRALIA, January 29, 1949) on his strikingly sane remarks concerning our attitude to the minor neurotics and psychotics. Many of these unfortunates have been so conditioned by their treatment at the hands of the psychiatrists that they consider their beloved "schizophrenia" a kind of lifelong gold pass which absolves them of the ordinary communal responsibilities of civilization and relieves them of the consequences of all types of antisocial behaviour.

A typical example is Mr. P., aged twenty-six years, who came to a busy surgery recently, settled himself comfortably in the chair, smoking a cigarette through fingers tobacco-stained to the wrist, and started thus: "Of course, Doc [to show his familiarity with things medical], I am a schizophrenic. I have been in Concord, Rockingham [*et cetera*, a long list follows] since 1940. My trouble is I am violent

and I can't sleep. I took fifteen grains of 'Nembutal' last night without effect. It's the same with 'Amytal', 'Soneryl', paraldehyde . . . [another list]." This goes on for an indefinite time limited only by the capacity for rudeness of the physician.

There is a rule of life which ultimately applies to all individuals, races and species. The sooner the schizophrenic individual and the schizophrenic nation appreciate this rule, the sooner shall world sanity and individual sanity prevail. The rule: "Work or starve."

Yours, etc.,

PHILIP STREITON.

Queenstown,
Tasmania,
February 3, 1949.

Obituary.

CHRISTOPHER ARTHUR GRIFFITH.

WE regret to announce the death of Dr. Christopher Arthur Griffith, which occurred on January 14, 1949, at Melbourne.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Fraser, Bruce St. Pierre, M.B., B.S., 1948 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

Heller, Edgar Herbert, M.B., B.S., 1948 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Pilcher, Russell Barton, M.B., B.S., 1948 (Univ. Adelaide), 39 Cross Roads, Kingswood, South Australia. Communications to Royal Adelaide Hospital.

Hobbs, Ian Harrold, M.B., B.S., 1948 (Univ. Adelaide), Payneham Road, Payneham, South Australia. Communications to Royal Adelaide Hospital.

Dunstone, Max Warwick, M.B., B.S., 1948 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Thomas, Brian Gordon, M.B., B.S., 1948 (Univ. Adelaide), Box 121, Port Lincoln, South Australia. Communications to Adelaide Children's Hospital, North Adelaide.

Steele, Ian McDonald, M.B., B.S., 1948 (Univ. Adelaide), 6 Moseley Street, Glenelg, South Australia. Communications to Royal Adelaide Hospital, Adelaide.

The undermentioned has applied for election as a member of the Tasmanian Branch of the British Medical Association:

Rose, Frederick Thomas, M.B., B.S., 1940 (Univ. Melbourne), 54 Gunn Street, Devonport.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Banfield, John Francis, M.B., B.S., 1948 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Elliott, Jack Raymond, M.B., 1940 (Univ. Sydney), Newcastle Hospital, Newcastle.

Grant, Alexander John, M.B., 1947 (Univ. Sydney), 72 Norfolk Road, Epping.

Lamond, Paul Kenneth, M.B., B.S., 1948 (Univ. Sydney), 15 Bayview Crescent, Annandale.

Lyle, Jean Frances, M.B., B.S., 1948 (Univ. Sydney), Eastern Suburbs Hospital, Bondi Junction.

McReady, Philip Archibald, M.B., B.S., 1946 (Univ. Sydney), Box 4380, G.P.O., Sydney.

Manchester, Edward Gordon Haig, M.B., B.S., 1948 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Mason, John Herbert, M.B., B.S., 1948 (Univ. Sydney), Sydney Hospital, Sydney.

Paterson, David Bruce, M.B., B.S., 1948 (Univ. Sydney), Parramatta District Hospital, Parramatta.

Piper, Douglas William, M.B., B.S., 1948 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Purnell, John Cecil, M.B., B.S., 1948 (Univ. Sydney), Western Suburbs Hospital, Croydon.

Smith, Clarice May, M.B., B.S., 1948 (Univ. Sydney), Provisional Registration, Eastern Suburbs Hospital, Bondi Junction.

Walter, Clement Jack, M.B., B.S., 1947 (Univ. Sydney), 442 Mowbray Road, Lane Cove.

Diary for the Month.

MARCH 1.—Federal Council of the B.M.A. in Australia: Melbourne.
MARCH 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MARCH 2.—Western Australian Branch, B.M.A.: Council Meeting.
MARCH 2.—Victorian Branch, B.M.A.: Branch Meeting.
MARCH 3.—New South Wales Branch, B.M.A.: Special Groups Committee.
MARCH 3.—South Australian Branch, B.M.A.: Council Meeting.
MARCH 4.—Queensland Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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